THE SURGICAL CLINICS OF NORTH AMERICA

AUGUST 1979

PERIPHERAL VASCULAR SURGERY

CORNELL UNIVERSITY
MEDICAL COLLEGE
LIBRARY

Digitized by the Internet Archive
in 2023 with funding from
Kahle/Austin Foundation

https://archive.org/details/surgicalclinicso0059unse

THE SURGICAL CLINICS OF NORTH AMERICA

VOLUME 59 / NUMBER 4
AUGUST 1979

SYMPOSIUM ON
PERIPHERAL VASCULAR SURGERY

John A. Mannick, M.D., *Guest Editor*

W. B. SAUNDERS COMPANY — Philadelphia · London · Toronto

W. B. SAUNDERS COMPANY: West Washington Square
Philadelphia, Pennsylvania 19105

1 St. Anne's Road
Eastbourne, East Sussex BN21 3UN, England

1 Goldthorne Avenue
Toronto, Ontario M8Z 5T9, Canada

Library of Congress Cataloging in Publication Data
Main entry under title:

Symposium on peripheral vascular surgery.

(The Surgical clinics of North America; v. 59, no. 4)
1. Blood-vessels—Surgery. I. Mannick, John A., 1928– II. Series.
[DNLM: 1. Vascular surgery. W1 SU7641L v. 59 no. 4 / WG170 S989]
RD34.S85 vol. 59, no. 4 [RD598.5] 617'.008s
[617'.413] 79-12203

The Surgical Clinics are also published in translated editions by the following:

Spanish	Nueva Editorial Interamericana, S.A. de C.V., Cedro 512, Apartado 26370, Mexico 4, D.F., Mexico
Italian	Piccin Editore, Via Porciglia 10, 35100 Padua, Italy
Turkish	Guven Bookstore, Mudafaa Cad. 16–B, P.O. Box 145, Kizilay, Ankara, Turkey
Portuguese	Editora Interamericana do Brazil, Ltda. Av. Venezuela, 131 2. Andar Rio de Janeiro, Brazil
Greek	Mr. Tassos Psaropoulos, Anglo-Hellenic Agency, 5 Koumpari Street, Athens 138, Greece

THE SURGICAL CLINICS OF NORTH AMERICA
August 1979 Volume 59—Number 4

© 1979 by W. B. Saunders Company. Copyright under the International Copyright Union. All rights reserved. This publication is protected by copyright. No part of it may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without written permission from the publisher. Made in the United States of America.

The following information is published in accordance with the requirements of the United States Postal Code.

The Surgical Clinics of North America (USPS 529–800) is published every other month by W. B. Saunders Company, West Washington Square, Philadelphia, Pennsylvania 19105, at Hampton Road, Cherry Hill, New Jersey 08002. Subscription price is $30.00 per year. Second-class postage paid at Cherry Hill, New Jersey. POSTMASTER: Send address changes to W. B. Saunders Company, West Washington Square, Phila., PA 19105

This issue is Volume 59, Number 4.

The editor of this publication is Linda C. Belfus, W. B. Saunders Company, West Washington Square, Philadelphia, Pennsylvania 19105.

Library of Congress catalog card number 39-19714.

Contributors

JOHN J. BERGAN, M.D., Magerstadt Professor of Surgery, and Chief, Division of Vascular Surgery, Northwestern University Medical School, Chicago, Illinois

VICTOR M. BERNHARD, M.D., Professor of Surgery, Medical College of Wisconsin, Milwaukee, Wisconsin

F. WILLIAM BLAISDELL, M.D., Professor of Surgery and Chairman, Department of General Surgery, University of California–Davis School of Medicine, Davis, California

DAVID C. BREWSTER, M.D., Assistant Professor of Surgery, Harvard Medical School; Assistant in Surgery, Massachusetts General Hospital, Boston, Massachusetts

SEBASTIAN CONTI, M.D., Assistant Professor of Surgery, Department of General Surgery, University of California–Davis School of Medicine, Davis, California

CHILTON CRANE, M.D., Emeritus Professor of Surgery, Peter Bent Brigham Hospital, Harvard Medical School, Boston; Chief of Surgery, Veterans Administration Hospital, West Roxbury, Massachusetts

E. STANLEY CRAWFORD, M.D., Professor of Surgery, Baylor College of Medicine and The Methodist Hospital, Houston, Texas

R. CLEMENT DARLING, M.D., Associate Clinical Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital, Boston, Massachusetts

R. CLEMENT DARLING, III, B.S., Surgical Technician, Massachusetts General Hospital, Boston, Massachusetts

DAVID J. EFFENEY, M.D., Assistant Professor of Surgery, University of California, San Francisco; Staff Vascular Surgeon, Veterans Administration Hospital, San Francisco, California

WILLIAM R. FLINN, M.D., Fellow in Peripheral Vascular Surgery, Northwestern University Medical School, Chicago, Illinois

THOMAS J. FOGARTY, M.D., Staff Surgeon, Sequoia Hospital, Redwood City, California; Private Practice, Palo Alto, California

WILLIAM J. FRY, M.D., Lee Hudson-Robert Penn Professor and Chairman, Department of Surgery, University of Texas Health Science Center at Dallas; Chief of Surgery, Parkland Memorial Hospital, Dallas, Texas

JOHN W. HALLETT, JR., M.D., Research Fellow, Harvard Medical School; Research and Clinical Fellow, Massachusetts General Hospital, Boston, Massachusetts

JAMES W. HOLCROFT, M.D., Assistant Professor of Surgery, Department of General Surgery, University of California–Davis School of Medicine, Davis, California

ANTHONY J. IMPARATO, M.D., Professor of Surgery, Department of Surgery, New York University Medical School; Attending Surgeon, New York University Hospital; Visiting Surgeon, Bellevue Hospital, New York, New York; Consulting Surgeon, Patterson General Hospital, Patterson, New Jersey; Norwalk Hospital, Norwalk, Connecticut; Veterans Administration Hospital, New York, New York.

JOHN A. MANNICK, M.D., Moseley Professor of Surgery, Harvard Medical School, Boston; Surgeon-in-Chief, Peter Bent Brigham Hospital, Boston, Massachusetts

CORNELIUS OLCOTT, IV, M.D., Assistant Clinical Professor of Surgery, University of California, San Francisco, California

ARTHUR E. PALAMARA, M.D., Cardiovascular Surgical Fellow, Baylor College of Medicine and The Methodist Hospital, Houston, Texas

LUIS A. QUERAL, M.D., Fellow in Peripheral Vascular Surgery, Northwestern University Medical School, Chicago, Illinois

JOHN O. F. ROEHM, JR., M.D., Clinical Associate Professor of Radiology, Baylor College of Medicine and The Methodist Hospital, Houston, Texas

SALWA A. SALEH, M.D., Clinical Instructor in Anesthesiology, Baylor College of Medicine and The Methodist Hospital, Houston, Texas

RONALD J. STONEY, M.D., Associate Professor of Surgery, University of California, San Francisco, California

D. E. STRANDNESS, JR., M.D., Professor of Surgery, University of Washington School of Medicine, Seattle, Washington

D. EMERICK SZILAGYI, M.D., Department of Surgery, Henry Ford Hospital, Detroit, Michigan

C. M. TALKINGTON, M.D., Associate Attending Surgeon, Baylor University Medical Center, Dallas, Texas; Clinical Assistant Professor of Surgery, University of Texas Southwestern Medical School, Dallas, Texas

JESSE E. THOMPSON, M.D., Attending Surgeon and Director of Graduate Education in Peripheral Vascular Surgery, Baylor University Medical Center, Dallas; Clinical Professor of Surgery, University of Texas Southwestern Medical School, Dallas, Texas

EDWIN J. WYLIE, M.D., Professor and Vice-Chairman, Department of Surgery, University of California, San Francisco; Chief, Vascular Surgery Service, University of California, San Francisco, California

JAMES S. T. YAO, M.D., Associate Professor of Surgery, and Director, Blood Flow Laboratory, Northwestern University Medical School, Chicago, Illinois

Contents

Foreword **537**

John A. Mannick

Carotid Surgery for Cerebral Ischemia **539**

Jesse E. Thompson and C. M. Talkington

Changing concepts of the causes, diagnosis, and treatment of strokes have led to the use of endarterectomy as a preventive measure, particularly in patients with transient ischemic attacks. Selection of patients, and diagnostic and surgical techniques are discussed.

Renal Artery Reconstruction **555**

William J. Fry

A substantial number of hypertensive patients whose disease is caused by renal ischemia remain untreated because no easy screening technique is available. Pyelograms and arteriography may be used for diagnosis, and results of transaortal endarterectomy or aortorenal bypass using autogenous saphenous vein grafting have been excellent.

Aorto-Iliac Reconstruction **565**

R. Clement Darling, David C. Brewster, John W. Hallett, Jr., and R. Clement Darling, III

Chronic aorto-iliac disease is manifested by progressive claudication, ischemic pain, ulceration, or gangrene. In properly selected patients good results have been obtained with aorto-iliac reconstruction by endarterectomy or bypass grafting with Dacron prostheses.

Femoro-Popliteal and Femoro-Tibial Reconstructions 581

John A. Mannick

Femoro-popliteal and femoro-tibial reconstructions have hitherto shown poor results, leading to skepticism on the part of surgeons. Careful attention to technical detail and on-the-table angiography have improved the prognosis of these procedures in patients with disabling claudication or those in danger of major amputation.

Aortic Aneurysm: Current Status of Surgical Treatment 597

E. Stanley Crawford, Arthur E. Palamara, Salwa A. Saleh, and John O. F. Roehm, Jr.

All aneurysms of the aorta are now successfully treated by operation; however, the indications, objectives, and results of therapy vary with the type and location of the aneurysm. This report emphasizes current concepts and principles.

Visceral Artery Syndromes and Reconstructions 637

Ronald J. Stoney and Cornelius Olcott

Acute mesenteric infarction, particularly with acute thrombosis, has long been resistant to surgical treatment. Improved results are possible with the use of embolectomy and revascularization prior to resection of the bowel, although controversy remains about the use of this method in celiac compression syndrome.

Extraanatomic Bypass Grafts 649

James W. Holcroft, Sebastian Conti, and F. William Blaisdell

Extraanatomic arterial bypass grafting is used when risk of standard operations is high because of associated disease or technical problems, in patients with ischemic lower extremities as a result of severe pulmonary or cardiac disease. Results have been good with femoro-femoral or axillo-femoral bypass.

Management of Complications after Arterial Reconstruction 659

D. Emerick Szilagyi

Early complications such as hemorrhage, infection, thrombosis, and neurologic loss are rare. A meticulous follow-up of each patient can help to identify late complications such as graft occlusion, false aneurysm, infection, and recurrence of atherosclerosis at an early stage when secondary surgical intervention is most satisfactory.

Surgery of the Aortic Arch Branches and Vertebral Arteries 669

Edwin J. Wylie and David J. Effeney

Prevention of stroke and relief of disabling symptoms of hypoperfusion are the objectives of vascular reconstruction in the proximal brachiocephalic and vertebral arteries. Endarterectomy, graft replacement, or "in line" bypass grafts are generally preferable to more remote transcervical bypass operations.

The Role of Profundaplasty in Revascularization of the Lower Extremities 681

Victor M. Bernhard

The deep femoral artery becomes the most important collateral vessel in the ischemic lower extremity when the superficial femoral artery is occluded. Profundaplasty is an important adjunct to inflow procedures when the deep femoral artery is stenotic.

Management of Peripheral Arterial Aneurysms 693

Luis A. Queral, William R. Flinn, James S. T. Yao, and John J. Bergan

True atherosclerotic aneurysms of the common femoral artery are less common than iatrogenic, mycotic, or postreconstructive pseudoaneurysms, but the development of new noninvasive diagnostic measures may lessen the incidence of false aneurysms. Usual treatment is by replacement grafting or simple suture of the iatrogenic arterial defect.

The Use and Abuse of the Vascular Laboratory 707

D. E. Strandness, Jr.

Noninvasive testing methods for evaluation of arteriosclerosis obliterans, extracranial arterial disease, and acute venous thrombosis may be performed in the office with a pocket-sized Doppler instrument. For plethysmography and ultrasonic imaging, however, a hospital laboratory with well trained personnel is a necessity.

Lumbar Sympathectomy: Role in the Treatment of Occlusive Arterial Disease in the Lower Extremities 719

Anthony J. Imparato

The most striking effects of sympathectomy in relieving ischemic rest pain and ulcers of the feet are notice-

able in the immediate postoperative period, and amputation may be postponed in most patients without gangrene and who show a rich collateral circulation. However progressive arteriosclerosis and trauma to ischemic limbs compromise long-term results.

The Surgery of Varicose Veins 737

Chilton Crane

Functional failure of venous valves is the central pathologic fault in varicose veins. Stripping of the saphenous vein has given good results; sclerotherapy is less successful but is useful in some situations.

Management of Arterial Emboli 749

Thomas J. Fogarty

In the clinical emergency presented by acute arterial occlusion, embolization must be distinguished from thrombosis, which requires complicated and time-consuming surgical techniques. The inventor of the Fogarty balloon catheter for embolectomy explains its use in conjunction with care of the underlying cardiac disease in patients with arterial emboli.

Index 755

RECENT SYMPOSIA

December 1978
Burns

February 1979
Endocrine Surgery

April 1979
Surgical Aspects of Immunology

June 1979
Advances in Small Hospital Care

FORTHCOMING SYMPOSIA

October 1979
Morbid Obesity
Henry Buchwald, M.D., *Guest Editor*

December 1979
New Methods of Treatment of Gastrointestinal Disease
G. V. Brindley, Jr., M.D., *Guest Editor*

Foreword

This issue of the Surgical Clinics of North America has been assembled with the goal of providing practical information of use to the clinician, particularly the clinical surgeon, who is called upon to manage patients with peripheral vascular disease. We have not attempted to be comprehensive or exhaustive in covering the field of peripheral vascular disease, but have focused on a number of commonly encountered vascular problems, particularly those in which management is currently controversial.

We have also made no attempt to present a single point of view or philosophy in the suggested management of the problems discussed in this issue. Rather we have called upon recognized experts in the various areas chosen for discussion and have asked them to present their views on patient evaluation and operative and nonoperative therapy without editorial alteration.

We hope that the result will prove both stimulating and useful to readers of the Surgical Clinics.

JOHN A. MANNICK, M.D.
Guest Editor

Department of Surgery
Harvard Medical School
Peter Brent Brigham Hospital
721 Huntington Avenue
Boston, Massachusetts 02115

Carotid Surgery for Cerebral Ischemia

*Jesse E. Thompson, M.D.,**
and C. M. Talkington, M.D.†

Stroke ranks third as a cause of death in the United States, surpassed only by heart disease and cancer. It is estimated that approximately 350,000 Americans suffer new strokes each year. One-third of those with acute thrombotic strokes die within 30 days of onset, and 200,000 persons in the United States die yearly. There are alive today in this country more than two million people who have had strokes. The social and economic consequences to the nation and to the families of these individuals are incalculable.

Changing concepts of the etiology, diagnosis, and treatment of strokes in the past two decades have been responsible for a widespread renewal of interest. Increasing awareness of the extracranial location and segmental nature of atherosclerotic occlusive lesions in a large proportion of patients with cerebrovascular insufficiency has been followed by the development and employment of appropriate vascular surgical techniques for removing or bypassing the offending plaques.[4, 18] It is now estimated that 75 per cent of patients with ischemic stroke syndromes have at least one obstructive lesion at a surgically accessible site.[8] As many as 40 per cent may have the principal occlusions confined to the extracranial vasculature. It is, therefore, technically feasible to increase cerebral blood flow or remove sources of emboli by surgical means.

More than three-fourths of the patients who suffer strokes have warning symptoms in the form of transient ischemic attacks. Likewise, 30 to 35 per cent of untreated patients with transient ischemic attacks develop frank strokes if followed three to five years or longer.[17]

A significant advance has been the recognition of amaurosis fugax and other eye symptoms as manifestations of carotid artery disease, from either emboli or reduction in blood flow. Amaurosis fugax is not a benign condition. It results in unilateral total blindness in a significant

*Attending Surgeon and Director of Graduate Education in Peripheral Vascular Surgery, Baylor University Medical Center, Dallas, Texas; Clinical Professor of Surgery, University of Texas Southwestern Medical School, Dallas, Texas

†Associate Attending Surgeon, Baylor University Medical Center, Dallas, Texas; Clinical Assistant Professor of Surgery, University of Texas Southwestern Medical School, Dallas, Texas

number of cases if untreated, and many patients go on to develop paralytic strokes.

Symptoms of cerebrovascular insufficiency result from two basic mechanisms: embolization into the brain of platelet aggregations or debris, or a reduction in cerebral blood flow. The natural history of ischemic thrombotic stroke due to extracranial lesions begins when a plaque forms at the common carotid bifurcation. Its first physical manifestation may be an asymptomatic bruit. Frequently the plaque becomes ulcerated and necrotic with deposition of platelets and thrombi on the ulcerated areas which may embolize to cause transient ischemic attacks or frank strokes. As a plaque enlarges and the size of the lumen diminishes, blood flow decreases. The final episode is thrombosis with complete occlusion. Hopefully the first symptom is a transient ischemic attack, but it may be a hemiplegic stroke. Thrombosis of the internal carotid artery usually involves its entire extracranial extent, which is associated with acute frank hemiplegic stroke in a significant proportion of cases. It is, therefore, pertinent to consider methods for prevention of stroke based on the natural history of the disease.

SIGNIFICANCE OF STENOTIC LESIONS

The question arises as to the functional significance of occlusive lesions seen on the arteriograms of the extracranial vessels supplying the brain. A lesion which is significant on x-ray examination may not be accompanied by clinical manifestations. Most significant carotid lesions show a reduction in diameter of the internal carotid artery of 40 to 50 per cent or more as measured on the arteriogram. A 50 per cent reduction in diameter results in a 75 per cent reduction in cross-sectional area, at which point significant pressure gradients begin to occur and blood flow begins to fall precipitously. A lumen of 1.0 mm in diameter or less always causes a significant pressure gradient and a reduction in flow in the internal carotid artery.[6] Stenoses of a lesser degree are of greater significance if the opposite carotid or the vertebral arteries are also compromised, or if multiple stenotic lesions are present. A stenosis of any degree may be significant if its appearance suggests the deposition of platelet thrombi or the presence of an ulcerated plaque, which may be sources of cerebral emboli.

CLASSIFICATION OF PATIENTS

The clinical syndromes of cerebrovascular insufficiency vary from a few minor symptoms to catastrophic stroke with paralysis and coma. Manifestations may be related to the carotid system, vertebral-basilar system, or both. The most common extracranial occlusive lesions amenable to surgical therapy are found in the carotid system.

It is important to classify patients into specific clinical categories based on the presenting neurologic status. Only in this way can proper

therapy be selected and results of different methods of treatment within the same category be compared. Prognosis for the individual patient also varies with the clinical classification, that is, worst for frank strokes and best for patients with asymptomatic bruits.

Several classifications of cerebrovascular insufficiency have previously been described. For purposes of surgical considerations related to carotid endarterectomy, we have chosen the following four clinical groups.[15] These are frank strokes: patients with neurologic deficits, including acute profound strokes, progressing strokes, recent and old completed strokes, fluctuating deficits, and mild neurologic deficits of more than 24 hours' duration; transient cerebral ischemia: patients with focal attacks of neurologic dysfunction and transient symptoms of generalized cerebral ischemia lasting minutes or hours, but without residual neurologic deficit at 24 hours; chronic cerebral ischemia: patients with obvious cerebrovascular insufficiency having loss of memory, impaired mentation, or overt motor or mental deterioration; and asymptomatic carotid bruits: patients without neurologic symptoms but with cervical bruits arising from occlusive carotid plaques demonstrated by arteriography. Over the years this classification has proved to be satisfactory for assessing surgical indications and evaluating results of operation. In addition to a clinical classification, anatomic classification of occlusions by accurate arteriography is essential.

INDICATIONS FOR AND TIMING OF CAROTID ENDARTERECTOMY

Clinical considerations in each of the four categories of patients determine the indications and contraindications for carotid endarterectomy. Indications for operation are listed in Table 1. The principal indication is transient cerebral ischemia since strokes can be prevented and troublesome episodes of ischemia are largely abolished. Patients with recent mild stable strokes or those with old strokes who develop new symptoms may also be candidates. Operation is contraindicated for cervical stenoses in the presence of severe intracranial disease.

Table 1. *Indications for Carotid Endarterectomy in Cerebrovascular Insufficiency*

Indications
Transient cerebral ischemia
Stable strokes, selected
Asymptomatic bruits, selected
Chronic cerebral ischemia, selected
Contraindications
Acute profound strokes
Progressing strokes
Severe intracranial disease
Other severe generalized disorders (e.g., cancer)

Table 2. *Timing of Carotid Endarterectomy for Cerebrovascular Insufficiency*

Elective operation
Stable stroke, recent or old
Transient cerebral ischemia
Asymptomatic bruit
Chronic cerebral ischemia (rare)
Delayed operation (days to weeks)
Mild stroke
Fluctuating stroke
Emergency operation (rarely necessary)
Frank stroke
Disappearance of bruit
Slowly worsening
Fluctuating
Transient cerebral ischemia
Severe stenosis, especially if bilateral
Disappearance of bruit
Carotid thrombosis immediately following arteriography or endarterectomy

Mild intracranial disease, however, is not a contraindication for operation. Age itself is not necessarily a contraindication to operation when the patient's general condition otherwise does not pose an undue hazard.

Indication for operation is also based on a critical evaluation of operative risk, since a high proportion of these patients have hypertension and generalized atherosclerosis, especially coronary disease. Javid et al.[10] have applied risk factors specifically to patients with asymptomatic bruits and believe endarterectomy is inadvisable in hypertensive patients over the age of 65 with a history of myocardial infarction.

Table 2 summarizes the appropriate timing of operation for various clinical categories. The procedure may be carried out at a time of election, delayed from several days to several weeks, or be done as an emergency. Although delayed operation is an important principle in stroke surgery, emergency operation is not often indicated. There is considerable difference of opinion regarding operation for acute carotid thrombosis following arteriography or endarterectomy. If operation can be performed immediately (one to two hours), it is our opinion that this is a justifiable procedure.

Likewise, controversy has arisen over the advisability of performing emergency endarterectomy on patients with unstable or fluctuating deficits and with crescendo transient ischemic attacks. Goldstone and Moore[7] have advocated an aggressive diagnostic and therapeutic approach. In our experience, management of these patients is fraught with difficulty. In the absence of completely reliable methods to distinguish between cerebral infarction and reversible ischemia without infarction, the results of therapy, whether surgical or medical, are un-

predictable. In general, if the deficit is mild and the lesion severe, emergency operation may be justified. Best results are usually obtained, however, if the patient's situation can be stabilized before operation is performed.

Patients with significant bilateral lesions should have bilateral endarterectomies, but in separate stages, at least one to two weeks apart or even longer. A number of strokes have occurred in patients with previous unilateral operations whose unoperated lesions were being followed.[15] Bilateral operation in a single stage is inadvisable because of the complications which may ensue, including respiratory difficulties, postoperative hypertension, and aggravation of neurologic deficits.

Occasionally one finds patients in whom occlusive lesions are located in the paradoxical carotid artery relative to the neurologic findings. Removal of these "inappropriate" lesions results in a clinical response to be expected had they been located on the appropriate side. This observation is in keeping with the concept of total cerebral blood flow on the basis of collateral circulation, which gives one a sound basis for recommending endarterectomy of "inappropriate" lesions.

DIAGNOSTIC STUDIES

Arteriography

Arteriography remains the definitive diagnostic maneuver. Four-vessel studies should be considered in every case but need not be routinely carried out unless indicated. Bilateral carotid arteriograms should be done routinely with serial films of the head and neck showing intracranial and extracranial vessels in both anteroposterior and lateral planes. If clinical findings are suggestive of vertebral-basilar as well as carotid insufficiency, one must perform vertebral studies.

Many satisfactory techniques for arteriography are now available. Direct carotid puncture in the neck may be used together with a percutaneous retrograde brachial technique to visualize both carotid and vertebral-basilar systems. Retrograde catheter techniques through the axillary or femoral arteries, utilizing local anesthesia, are being employed with increasing frequency, not only to visualize the aortic arch vessels but also to study selectively the extracranial carotid and vertebral arteries and the intracranial vasculature.

Radionuclide scans and, more recently, computed tomographic scans have been used in the investigation of stroke syndromes but their role is quite limited. There is little reason to perform these tests in patients with transient ischemic attacks, the principal indication for carotid surgery. Scans have been used in attempts to define the blood-brain barrier as a guide in timing surgical revascularization, but most surgeons have applied clinical criteria to determine operability, using the brain scan as an adjunct. The scans are most useful in demonstrating infarcts and mass lesions.

Noninvasive Studies

Although cerebral arteriography has become progressively safer, it still poses certain hazards.[11] Therefore, noninvasive methods of study have been sought in attempts to avoid unnecessary arteriograms in puzzling diagnostic situations and in the management of asymptomatic carotid bruit. This field is expanding rapidly as numerous noninvasive techniques are being devised and evaluated.[17]

At present writing, one of the most commonly employed noninvasive examinations is oculoplethysmography (OPG), combined with carotid phonoangiography (CPA), as reported by Kartchner et al.[11]

Our experience with the OPG-Kartchner apparatus, with which we have performed more than 5000 examinations, shows 90 per cent correlation with arteriograms. The instrument has been quite useful as a screening device for asymptomatic patients with carotid bruits and is singularly free of complications.

Directional Doppler studies of supraorbital blood flow also give helpful information. Normally, blood flows from the ophthalmic artery into the supraorbital artery and then by way of anastomotic branches into the superficial temporal branch of the external carotid artery. If stenosis or occlusion of the internal carotid artery exists, the direction of flow in the supraorbital artery may be reversed, which can be detected by ophthalmosonometry. The test correlates with arteriograms in about 80 per cent of cases. Compression of the facial, infraorbital, and superficial temporal arteries increases the accuracy of the examination. The combination of the above two methods yields a screening accuracy of greater than 90 per cent.

Ultrasonic scanning and Doppler imagery mapping of the cervical carotid artery are still in the developmental stage.[1] With perfection of the technology, such procedures promise to be extremely valuable, refining the indications for arteriography. They should be particularly useful in evaluating the asymptomatic bruit.

TECHNIQUE OF CAROTID ENDARTERECTOMY

The most common operation used in the treatment of cerebrovascular insufficiency is carotid endarterectomy. Technical considerations are of the utmost importance in this operation since the limits of tolerance to temporary occlusion of the blood supply to the brain may be quite narrow. Safety factors must be employed which will eliminate in most instances the occurrence or aggravation of neurologic deficits. Any method of carotid endarterectomy should carry low mortality, few complications, satisfactory immediate and long-term anatomical results, and good functional results relative to cerebrovascular insufficiency. Operation is usually performed under light general anesthesia. Figures 1 through 6 depict details of the technique. We use a shunt routinely, as illustrated.

Regional and systemic heparinization are used during endarterectomy. Postoperative heparinization is not employed. Adequate levels of

blood pressure must be maintained during and immediately after operation with 500 to 1000 ml of lactated Ringer's solution or with vasopressors.

Complications

Complications associated with carotid endarterectomy are listed in Table 3. With meticulous attention to the details of intraoperative and postoperative management, the actual incidence of complications is quite low, about 2 to 4 per cent for neurologic deficits and no more than 4 per cent for the remainder.

The most serious complication is the occurrence or aggravation of neurologic deficits, caused by embolization or cerebral ischemia. The former is caused by excessive manipulation of the artery, while the latter may result from hypotension, intracerebral thrombosis, or inadequate cerebral protection.

Although some surgeons employ selective shunting based on stump pressure measurements or electroencephalographic changes for cerebral protection,[6, 9] and others state they rarely or never use a shunt,[2] we have found routine shunting to be safest and most satisfactory when general anesthesia is used.

Severe hypertension is not uncommon postoperatively in patients with preexisting hypertension and must be treated aggressively to prevent hematomas, disruption of the reconstruction, and cerebral hemorrhage and edema. If blood pressure rises above 200 mm Hg, one should immediately employ an intravenous drip of 500 mg of Arfonad in 500 ml of 5 per cent dextrose in water, which lowers pressure within minutes. Intermittent injections or a properly titrated drip keeps the pressure at appropriate levels until spontaneous resolution occurs or other longer acting hypotensive agents take effect. Sodium nitroprusside (Nipride) solution, 50 mg in 500 to 1000 ml of 5 per cent dextrose in water, may also be administered by intravenous drip as a rapidly acting antihypertensive agent. Based on clinical experience, Arfonad is our preferred drug.

RESULTS OF ENDARTERECTOMY

The purpose of carotid endarterectomy is to remove occlusive plaques. By doing so, one eliminates sources of cerebral emboli and increases cerebral blood flow in an effort to relieve symptoms of cerebrovascular insufficiency, improve neurologic function, prevent strokes, and increase long-term survival.

Anatomic Restoration

Successful anatomic restoration of blood flow by operation may be accomplished in more than 98 per cent of patients with partially occluded arteries. Arteries reconstructed by endarterectomy remain patent for many years with a patency rate approaching 95 per cent of those studied.[13] In our own series reoperation for symptomatic recur-

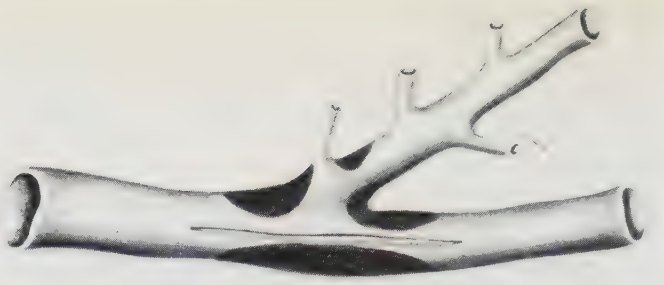


Figure 1. The technique of carotid endarterectomy utilizes a temporary inlying bypass shunt. An oblique incision is made in the neck along the anterior border of the sternocleidomastoid muscle, centered over the carotid bifurcation, curving medialward at its lower end, and curved slightly inferior to the lobe of the ear at the upper end, at the usual site of atherosclerotic plaque. The location of the linear arteriotomy is indicated from the common carotid into the internal carotid beyond the distal extent of the plaque so that its upper end is clearly visualized. (*From* Thompson, J. E.: Internal Carotid and Vertebral Occlusive Disease. *In* Hardy, J. D. (ed.): Rhoads Textbook of Surgery. Philadelphia, J. B. Lippincott Co., 1977, with permission.)

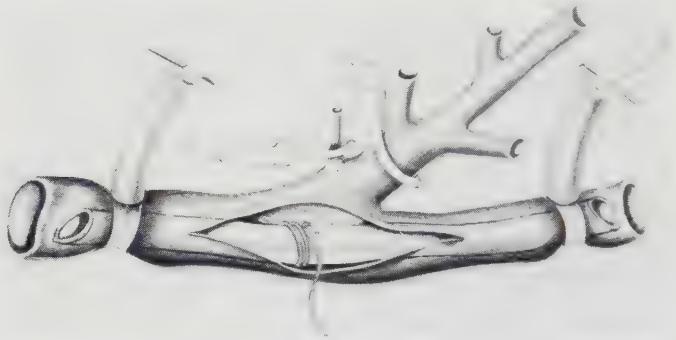


Figure 2. A 10 French plastic catheter 9 cm in length is first inserted into the distal internal carotid and allowed to back flow. The proximal end of the shunt is then placed into the common carotid lumen, and the umbilical tapes with rubber tourniquets are made snug. Cerebral blood flow is thus restored through the shunt, a step requiring approximately 60 seconds. (*From* Thompson, J. E.: Internal Carotid and Vertebral Occlusive Disease. *In* Hardy, J. D. (ed.): Rhoads Textbook of Surgery. Philadelphia, J. B. Lippincott Co., 1977, with permission.)

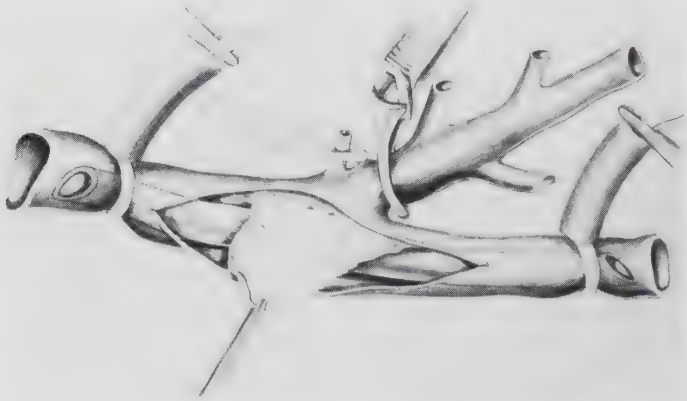


Figure 3. The appropriate plane is entered with a fine pointed clamp and endarterectomy of the common carotid, bifurcation, origin of external carotid, and the internal carotid is accomplished under full visualization. The distal end of the plaque in the internal carotid usually feathers off quite smoothly. If not, the distal intima may be secured with a few interrupted sutures of 6-0 polyester to prevent dissection. (*From* Thompson, J. E.: Internal Carotid and Vertebral Occlusive Disease. *In* Hardy, J. D. (ed.): Rhoads Textbook of Surgery. Philadelphia, J. B. Lippincott Co., 1977, with permission.)

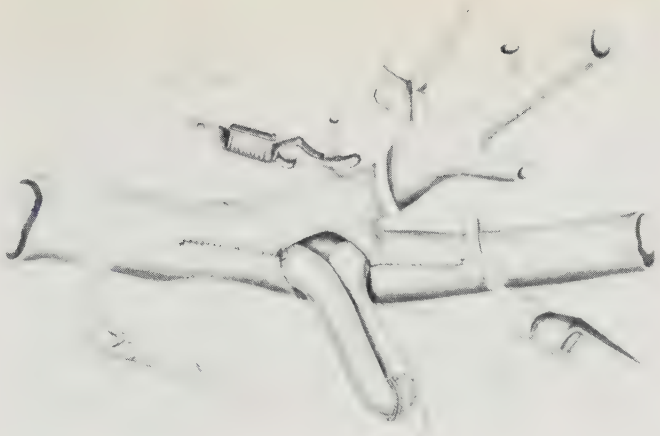


Figure 4. After flushing out the artery, the arteriotomy is closed with running sutures of 6-0 polyester beginning at each end. Immediately prior to placing the final three or four sutures, the common carotid and internal carotid artery are clamped and the shunt removed. The vessels are flushed and the final sutures placed and tied. Flow is restored, first into the external carotid and finally into the internal carotid. (*From* Thompson, J. E.: Internal Carotid and Vertebral Disease. *In* Hardy, J. D. (ed.): Rhoads Textbook of Surgery. Philadelphia, J. B. Lippincott Co., 1977, with permission.)

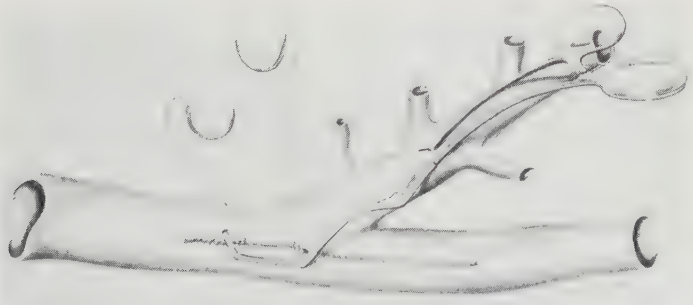


Figure 5. A variation on the technique of arteriotomy closure is shown in which a thin-blade partially occluding clamp is used to include the unfinished portion of the suture line after the shunt is removed. In this way cerebral blood flow is more quickly reestablished. The suture line is then completed. (*From* Thompson, J. E.: Internal Carotid and Vertebral Disease. *In* Hardy, J. D. (ed.): Rhoads Textbook of Surgery. Philadelphia, J. B. Lippincott Co., 1977, with permission.)

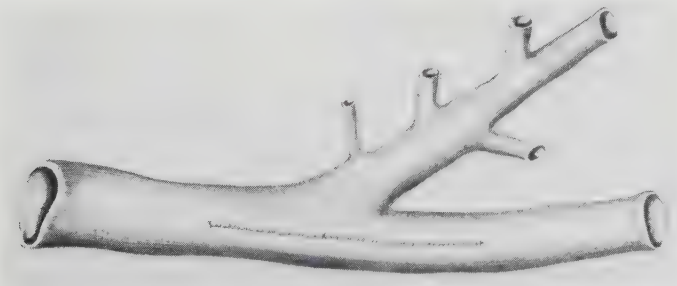


Figure 6. The artery is seen following final closure. A patch graft is rarely necessary unless the artery is unusually narrow. The wound in the neck is closed with two layers of fine cotton sutures about a small rubber drain which is removed 24 hours postoperatively. (*From* Thompson, J. E.: Internal Carotid and Vertebral Disease. *In* Hardy, J. D. (ed.): Rhoads Textbook of Surgery. Philadelphia, J. B. Lippincott Co., 1977, with permission.)

Table 3. *Complications of Carotid Endarterectomy*

Related to anesthesia, general or local
Cardiac problems
Airway problems
Hypotension
Related to cervical wound
Infection
Hematoma
Nerve pareses
Vagus
Hypoglossal
Marginal branch of facial
Parotitis
Tracheal obstruction
Related to carotid artery
Disruption
False aneurysms
Carotid-cavernous arteriovenous fistula
Infection of dacron graft
Production or aggravation of neurologic deficits
Intraoperative causes
Embolism
Cerebral ischemia
Postoperative causes
Thrombosis of endarterectomized segment
Hypotension
Intracerebral hemorrhage or edema
Hypertension
Miscellaneous
Postoperative headache
Cerebral edema

rent stenosis at the site of previous endarterectomy has been 0.6 per cent.

With totally occluded carotid arteries, however, restoration of patency by operation is low, being 40 per cent or less.

Operative Mortality

During a 20-year period the authors and their associates have performed 1286 carotid endarterectomies on 1022 patients with cerebrovascular insufficiency.[16] Operative mortality is shown in Table 4. In recent years general anesthesia has been employed, together with the routine use of a temporary inlying shunt for cerebral protection, and operation has not been performed on patients with acute and progressing strokes. Mortality has been halved in both frank stroke and transient ischemic attack. Operative mortality varies directly with the severity of the clinical classification of the patient.

Table 4. *Operative Mortality Following Carotid Endarterectomy (Total Experience 20 Years)*

Clinical Category	No. of Patients	No. of Operations	No. of Deaths	Patient Mortality (per cent)	Procedure Mortality (per cent)
Frank stroke	296	358	20	6.8	5.6
Transient ischemia	575	737	7	1.2	0.9
Chronic ischemia	19	24	0	0	0
Asymptomatic bruit	132	167	0	0	0
Total	1022	1286	27	2.6	2.1

Operation-Related Deficits

In our series of 516 operations on 395 patients with transient ischemic attacks and asymptomatic bruits, there were one death caused by stroke (0.2 per cent), four transient deficits (0.8 per cent), and seven permanent deficits (1.4 per cent).

Functional Results

Frank Strokes. Functional results for frank strokes are difficult to evaluate because of the wide variation in severity of neurologic deficits at onset and the natural history of improvement without treatment. These patients carry the highest neurologic risk factors. They should be selected for operation with great care after critical evaluation.

Transient Cerebral Ischemia. This should be the ideal stage for definitive therapy since no neurologic deficit is present.[5] In our series followed up to 13 years, 81 per cent of the survivors had no further transient ischemic attacks, 16 per cent were improved, 2 per cent were worse, and 1 per cent were normal or improved and then worsened with time. The total incidence of long-term strokes was 5.4 per cent.[15] Operative mortality and morbidity continue to improve with increasing experience. Mortality in transient ischemic attacks should approach 1 per cent, and the incidence of operation-related deficits should be no more than 2 to 4 per cent.

Total Carotid Occlusions and Acute Strokes. A considerable difference of opinion exists regarding operation on the totally occluded internal carotid artery. Success in reestablishment of blood flow is no higher than 40 per cent, depending upon the time interval from onset to operation.

Chronic total occlusion in the absence of symptoms is not an indication for operation. Chronic occlusion in the presence of symptoms may be justifiably operated upon if no other operable lesions exist, if bilateral total occlusions are present, if ipsilateral external carotid flow can be significantly improved, or if there is some indication on the arteriogram that successful restoration of flow is possible.

Acute total occlusion is a different matter entirely because of its association with acute frank hemiplegic stroke in a significant proportion of cases. Here the most important factor determining operability is the clinical nature of the stroke itself. Rarely, if a patient is in hospital when acute stroke with total occlusion occurs, and operation can be done within two hours, emergency endarterectomy may be justified. Otherwise studies should be postponed. Mortality rates up to 60 per cent follow operation for acute stroke in the early phase because of intracranial hemorrhage and cerebral edema which follow revascularization of acute cerebral infarction. It is now generally agreed that operation on patients with acute profound strokes, rapidly progressing strokes, and rapidly improving strokes is contraindicated in the acute stage; such patients should be allowed to stabilize two to six weeks or longer before studies are done and operation considered.

CAROTID STENOSIS WITH CONTRALATERAL OCCLUSION. Patients with unilateral carotid stenosis and contralateral occlusion present special problems since they may be quite symptomatic yet carry the highest risk of operation. Using a shunt routinely, we have performed 136 operations on the stenotic side without a death and with only one operation-related deficit. Functional results of endarterectomy in this group may be most dramatic and rewarding, provided the operation proceeds uneventfully.

ASYMPTOMATIC CAROTID BRUITS

The gratifying success of stroke prevention by endarterectomy in patients with transient ischemic attacks has prompted investigation into the operative management of asymptomatic patients with bruits arising from extracranial carotid stenosis.

In an attempt to study the natural history, we have followed up to 16 years, 270 patients with asymptomatic carotid bruits. Of these, 132 were subjected to endarterectomy primarily, whereas 138 were followed but not operated upon when the bruit was first detected. In the operated group, 167 endarterectomies were performed with no mortality. Two patients sustained transient neurologic deficits related to operation, which cleared completely, and two additional patients had permanent mild deficits, an incidence of 1.2 per cent. A continuous long-term follow-up study has been carried out on both groups of patients.[16] Long-term data on the neurologic status of the operated and nonoperated upon patients are shown in Table 5. The data appear to favor the operated patients as being at less risk for stroke or transient ischemic attacks. It does not appear unreasonable, therefore, to consider the asymptomatic bruit part of the total picture of cerebrovascular insufficiency rather than an innocent isolated finding on physical examination.

Corroborating our data, Cooperman et al.[3] have followed 60 patients with asymptomatic bruits from two to seven years. Twelve patients, or 20 per cent, had transient ischemic attacks, whereas five

Table 5. *Long-Term Follow-Up Data on Operated and Non-Operated Patients with Asymptomatic Carotid Bruits*

	ASYMP-TOMATIC	TRANSIENT ISCHEMIC ATTACKS	NONFATAL STROKE	FATAL STROKE	LENGTH OF FOLLOW-UP
Operated: 132 patients	120 90.9%	6 4.5%	3 2.3%	3 2.3%	6 to 184 months mean—55.1 months
Non-operated: 138 patients	77 55.8%	37 26.8%	21 15.2%	3 2.2%	To 180 months mean—45.5 months (ASX)
Significance	$p < 0.01$	$p < 0.01$	$p < 0.01$		

patients had nonfatal strokes (8.3 per cent) and four had fatal strokes (6.7 per cent) for a total stroke incidence of 15 per cent and an overall incidence of ischemic episodes of 35 per cent. Thirty-five per cent of the patients died of other causes. Thirty per cent were alive and neurologically asymptomatic at the end of the study.

Likewise, Kartchner and McRae, in an extensive screening survey with the OPG, found that patients with cervical bruits and negative findings on OPG had a 0.7 per cent incidence of carotid stroke, whereas those with pansystolic bruits and greater than 70 per cent stenosis by OPG had a 20.4 per cent incidence of carotid stroke, when followed up to 70 months.[12]

The questions that arise when one first hears a bruit in an asymptomatic patient are: does the murmur denote a significant lesion in the internal carotid artery, and how can one determine its significance without subjecting the patient to the risk of unnecessary arteriography? Safe, simple, and reliable noninvasive screening tests have been needed. Several such tests have already been developed while a number of others are under intensive investigation. With perfection of the technology in the future it is becoming possible with noninvasive examinations to assess both anatomic and functional hemodynamic components of the lesions responsible for bruits, thus making more precise the indications for arteriography, which remains at present the definitive diagnostic maneuver. Once arteriography has been accomplished, indications for endarterectomy are usually straightforward.

Management of the patient with an asymptomatic carotid bruit follows three steps: noninvasive screening, cerebral arteriography and carotid endarterectomy.

Noninvasive Screening

Noninvasive screening tests should be applied to all patients. At present we use OPG-CPA (Kartchner) and supraorbital Doppler studies.

Cerebral Arteriography

As cerebral arteriography has become increasingly safer with retrograde techniques and local anesthesia, it may be recommended more liberally than previously, but not routinely, in evaluating asymptomatic bruits of grade II intensity or greater. The overall general status of each patient should be considered very carefully. Arteriography should not be done if contraindications to endarterectomy already exist. It is not to be recommended if the bruit is soft and unilateral, if other conditions take priority over study of the bruit, or if the noninvasive tests are negative. It may be recommended if bruits are bilateral and harsh, in patients with known progressive atherosclerosis elsewhere, prior to other operations, when noninvasive tests are positive, and when one's best clinical judgment indicates that it is necessary. An experienced arteriographer should be available.

Carotid Endarterectomy

If the arteriograms show a significant atherosclerotic stenosis in the common or internal carotid artery, endarterectomy may be recommended. Specific indications include bilateral stenoses, unilateral stenosis with contralateral occlusion, stenosis in the artery to the dominant hemisphere, known progressive atherosclerosis elsewhere in the peripheral vasculature, especially in younger patients, contemplated major operation of another sort, particularly open-heart surgery, and a markedly ulcerated plaque.

Prophylactic carotid surgery should not be considered in the presence of multiple risk factors, such as a history of hypertension, myocardial infarction, and congestive heart failure in patients over 65. Since no unnecessary technical risks should be taken, appropriate measures for cerebral protection must be used during endarterectomy to avoid producing neurologic deficits. Our recommendation is the routine use of a temporary inlying shunt. Operative mortality should be below 1 per cent and complications no more than 2 per cent.

Role of the External Carotid Artery

The external carotid artery is an important source of intracranial collateral circulation in the presence of a stenosed or occluded internal carotid artery. During routine endarterectomy any plaques in the origin or accessible portions of the external carotid artery should be removed.

In the presence of symptomatic chronic total occlusion of the internal carotid artery with coexisting external carotid plaques, removal of the latter may be followed by improvement in symptoms of transient ischemic attack and amaurosis fugax. For many years we have operated on selected lesions, employing a shunt in the external carotid artery to protect the collateral circulation to the eye.

Recent hemodynamic studies have demonstrated that stump pressure in the internal carotid artery may be augmented when external carotid flow is restored in the presence of clamping of the internal carotid artery. This observation has important technical implications under special circumstances.

REFERENCES

1. Barnes, R. W., Bone, G. E., Reinertson, J., et al.: Noninvasive ultrasonic carotid angiography: Prospective validation by contrast arteriography. Surgery, *80*:328, 1976.
2. Baker, W. H., and Dorner, D. B.: Carotid endarterectomy: Is an indwelling shunt necessary? Surgery, *82*:321, 1977.
3. Cooperman, M., Martin, E. W., and Evans, W.: Significance of asymptomatic carotid bruits. Arch. Surg., *113*:1339, 1978.
4. DeBakey, M. E.: Patterns of atherosclerosis and rates of progression. Atherosclerosis Reviews, *3*:1, 1978.
5. DeWeese, J. A., Rob, C. G., Satran, R., et al.: Results of carotid endarterectomies for transient ischemic attacks — five years later. Ann. Surg., *178*:258, 1973.
6. Engell, H. C.: Studies in cerebral circulation. Bull. Am. Coll. Surg., *58*:7, 1973.
7. Goldstone, J., and Moore, W. S.: Emergency carotid artery surgery in neurologically unstable patients. Arch. Surg., *111*:1284, 1976.
8. Hass, W. K., Fields, W. S., North, R. R., et al.: Joint study of extracranial arterial occlusion. II. Arteriography, techniques, sites, and complications. J.A.M.A., *203*:961, 1968.
9. Hays, R. J., Levinson, S. A., and Wylie, E. J.: Intraoperative measurement of carotid back pressure as a guide to operative management for carotid endarterectomy. Surgery, *72*:953, 1972.
10. Javid, H., Ostermiller, W. E., Hengesh, J. W., et al.: Carotid endarterectomy for asymptomatic patients. Arch. Surg., *102*:389, 1971.
11. Kartchner, M. M., and McRae, L. P.: Noninvasive evaluation and management of the "asymptomatic" carotid bruit. Surgery, *82*:840, 1977.
12. Kartchner, M. M., and McRae, L. P.: Unpublished data, 1978.
13. Stoney, R. J., and String, S. T.: Recurrent carotid stenosis. Surgery, *80*:705, 1976.
14. Thompson, J. E.: Prevention of complications of cerebral arteriography and surgery. *In* Dale, W. A. (ed.): Management of Arterial Occlusive Disease. Chicago, Year Book Medical Publishers, Inc., 1971.
15. Thompson, J. E., Austin, D. J., and Patman, R. D.: Carotid endarterectomy for cerebrovascular insufficiency: Long-term results in 592 patients followed up to thirteen years. Ann. Surg., *172*:663, 1970.
16. Thompson, J. E., Patman, R. D., and Talkington, C. M.: Asymptomatic carotid bruit: Long-term outcome of patients having endarterectomy compared with unoperated controls. Ann. Surg., *188*:308, 1978.
17. Thompson, J. E., and Talkington, C. M.: Carotid endarterectomy: Surgical progress. Ann. Surg., *184*:1, 1976.
18. Wylie, E. J., and Ehrenfeld, W. K.: Extracranial Occlusive Cerebrovascular Disease. Philadelphia, W. B. Saunders Co., 1970.

Suite 505
3600 Gaston Avenue
Dallas, Texas 75246

Renal Artery Reconstruction

*William J. Fry, M.D.**

Since the correlation between hypertension and renal ischemia was demonstrated by Goldblatt et al., in 1934, only sporadic interest has been demonstrated in the literature.[8] Unfortunately, enthusiasm has remained largely with the vascular surgeon and not the primary care physician nor with the secondary care medical hypertension specialist.

There is no good estimate of the incidence of hypertension in the United States. Figures range from a low of five million to a high of as many as 22 million. Depending upon referral patterns and on location, the incidence of hypertension as a result of renal ischemia varies between 6 to a high of 37 per cent of all hypertensive patients. A practical figure would appear to be 15 per cent.[6]

No matter how conservative one is with figures, it is readily apparent that there are a substantial number of people in the United States with potentially remediable hypertension. These patients are not being seen and cared for by the vascular surgeon for several reasons. Large experiences with good results are not commonplace and are clustered in a few centers. The difficulty of making a diagnosis without using arteriographic techniques precludes large-scale screening programs. Thus, many patients carry an unnecessary burden of risk because of misconception, lack of awareness, or financial limitation.[10]

Because no easy screening technique is available, except for a high index of suspicion combined with mutual respect between internist and surgeon, care of patients must be a combined effort among all disciplines.

Adjunctive Diagnostic Procedures

The intravenous pyelogram is frequently used as a screening test.[6, 9, 12, 17] It is not a highly selective test for renovascular hypertension, being accurate in only 24 per cent of patients in the pediatric age group and in 47 per cent of patients in the young adult age group with

*Lee Hudson-Robert Penn Professor and Chairman, Department of Surgery, The University of Texas Health Science Center at Dallas; Chief of Surgery, Parkland Memorial Hospital, Dallas, Texas

fibrodysplastic disease. It is a more accurate method of determination in the atherosclerotic age group, being accurate in 73 per cent of patients with focal atherosclerotic disease, and in 71 per cent of patients with general atherosclerosis. It is important to point out that anyone with hypertension should have a screening intravenous pyelogram. While accuracy for determining hypertension is low, it does allow accurate delineation of the architecture of the kidney and helps to rule out neoplasms and other parenchymal diseases such as pyelonephritis, obstructive uropathy, and so forth.

Lesions of segmental renal arteries or long-standing renal artery stenosis are the main reasons for the inaccuracies of the intravenous pyelogram, as well as other adjunctive function tests. These lesions cannot be accurately delineated by the intravenous pyelogram because of their segmental nature or because of the increased flow of blood through collateral vessels into the renal parenchymal system. The intravenous pyelogram, then, is a measure only of gross changes in function and will not show subtle changes in blood flow or pressure that will produce hyperreninemia.

Renin determinations would seem to hold the most potential as a screening and selective test to determine the existence of renovascular hypertension. Many inconsistencies exist in the current literature regarding the usefulness of renin assays. There is great variability in the performance of the collection of the renin samples, and investigators measure renin levels after various methods of stimulation. This is further complicated because in the severe hypertensive who is on blocking agents for the control of elevated blood pressure, no accurate determination of renin production can be made. When it is feasible to prepare a patient by administering a natruretic drug, exercising the patient and withdrawing simultaneous samples with the patient in the upright position, renin determinations are accurate.

We have found that the renin ratio determination has not been as accurate as the use of the renal systemic renin indices as defined by individual renin activity minus the systemic activity divided by the systemic renin activity:[18]

$$\text{RSRI} = \frac{\text{individual renal renin activity} - \text{systemic renin activity}^{18}}{\text{systemic renin activity}}$$

Utilizing these indices it is possible, when the patient is adequately prepared, to determine with high reliability the result of a technically successful operation. Indices in the renal system of renin also indicate whether a patient will be cured or merely improved. If the contralateral kidney is not suppressed in its renin production by the diseased side, enough renin will be produced to preclude cure of the patient; and will result in improvement only.

The utilization of an competitive inhibitor of angiotensin II (saralasin) would seem to be a good method of screening patients to test whether high renin production is the cause of hypertension.[1] The same pitfalls are seen with this drug as are seen in renin determinations. In those patients with severe hypertension, particularly those on propran-

olol, there will be no response with saralasin infusion. In addition, some hyperrenin-secreting patients have no readily explained etiologic factor accounting for the excess renin production. The use of saralasin in the sodium-depleted patient, however, holds promise, as pointed out by Marks et al.[11]

We have not used differential renal function studies as delineated by Stamey, Howard et al, since their accuracy has not been as good as renin determinations.[11, 9] In addition to this, renal function studies are time-consuming and have a high incidence of associated morbidity. Accuracy is again clouded in patients with segmental disease or with the development of prodigious collateral circulation.[4] As with the other screening tests, studies of renal function rely on gross changes and cannot, by their nature, delineate all areas of ischemia or changes in pulse-pressure relationships. Dean and Foster report a 32 per cent false normal rate using the Howard test and a 58 per cent false negative rate using Stamey's modification.[6] This should preclude the use of these examinations, even in combination as recommended by Dean and Foster, since an eight to ten per cent false negative rate remains in patients subsequently operated upon and cured of their hypertension.

Many isotopes have been utilized as a screening test for patients with hypertension in an attempt to determine blood flow. At best, there is a 14 per cent false negative rate in patients with renal artery stenoses, and the tests are of no value in patients with bilateral disease. New techniques are being developed which allow assessment of relative renal blood flow and this promises to be useful in the future. Scans may be helpful in the immediate postoperative period if there is a question regarding blood flow after an angioplastic procedure.

Arteriography

Arteriography remains the most reliable technique for determining significant renal artery disease. The accuracy of arteriography has improved remarkably with the use of selective catheter placement, multiple oblique views, and, recently, vasoactive drugs to better demonstrate collateral blood supply around a partially occluding lesion in the renal vasculature.[3] All patients should have a flush abdominal aortogram not only to delineate general architecture of the renal arteries but to show the visceral vessels and the abdominal aorta. Simultaneous existence of splenic artery aneurysms and occlusive disease within the visceral vessels and the aortoiliac system is commonplace and adds significantly to preoperative planning before a patient is subjected to an angioplastic procedure.[13]

Careful views of the aorta and iliac vessels are of great importance, particularly in the atherosclerotic patient, to determine the feasibility of either endarterectomy or bypass. Careful selective views, with multiple projections and preferably magnification, should always be obtained when assessing the renal vascular architecture. Multiple lesions, which are common with fibrodysplastic disease, may be hidden by the course of the vessel and may be seen only with careful attention to an oblique projection. Intraparenchymal views, preferably

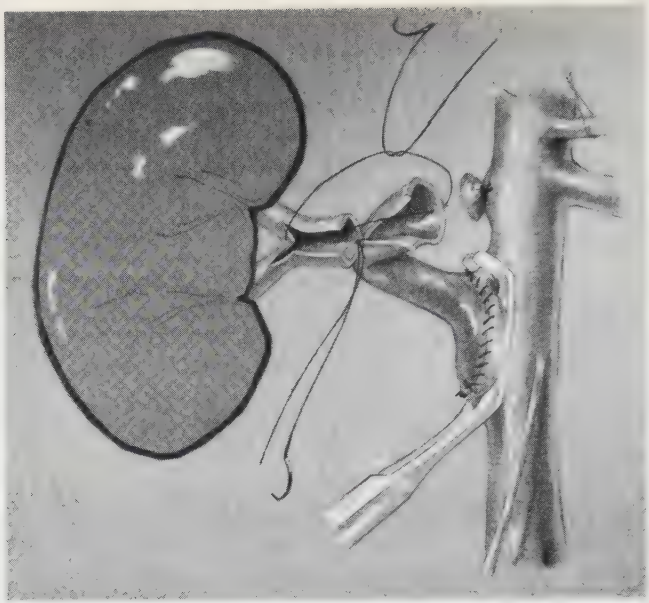


Figure 1. The technique of distal end-to-end anastomosis is shown as it is used in renal revascularization.

magnified, are important to demonstrate small areas of infarction or microaneurysms, such as those seen in some forms of collagen disease. It is apparent that the care and judgment utilized in the performance of renal arteriography expand the ability of the surgeon and significantly aid in forecasting the result of a successful angioplastic procedure.

Operative Technique

We have preferred aortorenal bypass utilizing an autogenous saphenous vein as the procedure of choice in revascularization procedures.[5] It is important to point out that autogenous tissue has many advantages over the synthetics, either knitted Dacron or expanded Teflon (Impra) grafts.[5, 20, 22, 23, 16] Autogenous tissue is pliable, easy to tailor, and is amenable to fine suture technique, whereas the synthetic grafts now available preclude accurate anastomoses to vessels as small as one millimeter. We have felt that the distal anastomosis should be done in an end-to-end fashion rather than an end-to-side manner (Fig. 1). The flow characteristics of an end-to-end anastomosis preclude eddy currents which produce turbulent flow and may cause medial changes in the vessel wall. We have observed propagation of thrombus in end-to-side anastomoses, occluding the ostium of the vein graft. An additional technical advantage is found in the end-to-end anastomosis in that the graft will lie easily, and generally there is no problem with tension. In the end-to-side anastomosis, the judgment of graft length is difficult and thus one may produce bowing and narrow-

ing of the renal artery if the graft is too short or too tight. This is particularly true if a plastic prosthesis is used.

As delineated in the diagram, the distal anastomosis should be done with an ostium roughly three times as large as the lumen of the renal artery (Fig. 2). This is accomplished by spatulating both the renal artery and the vein graft. Utilizing this technique, we have seen no strictures of the suture line. As healing occurs and the cicatrix forms, any contraction is eccentric and will not cause narrowing of the lumen of the renal artery. This is particularly important when one considers the pediatric age group where growth will take place. We have not seen any suture line strictures in any of our pediatric patients during rapid growth associated with puberty.[7]

Early in our experience with autogenous saphenous vein, we found that aneurysms formed in six per cent of our bypass grafts.[15] We think that this was a result of inadequate harvesting and preservation of vein grafts. It is important to harvest veins only after dissection around the kidney and aorta has been completed. In this way the vein graft is without a blood supply for the shortest possible time. In addition, we have immediately submersed the vein graft in cold, heparinized blood. This has been shown in the laboratory to preserve the integrity of the media so that minimal changes take place within the saphenous vein.[19] The side clamp should be left on the aorta until the

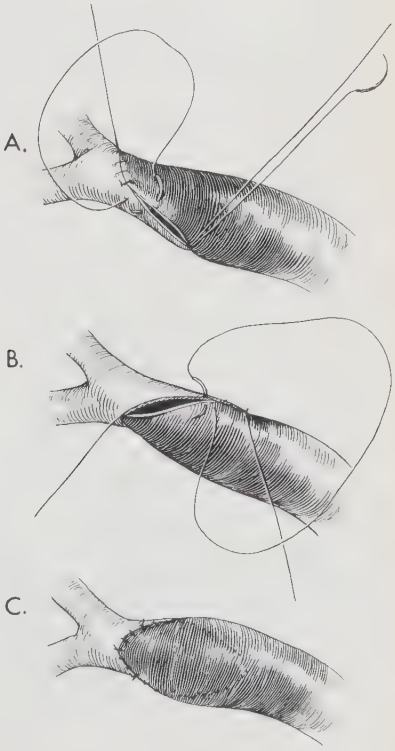


Figure 2. Spatulation of the end-to-end anastomosis eliminates stricture at the suture line.

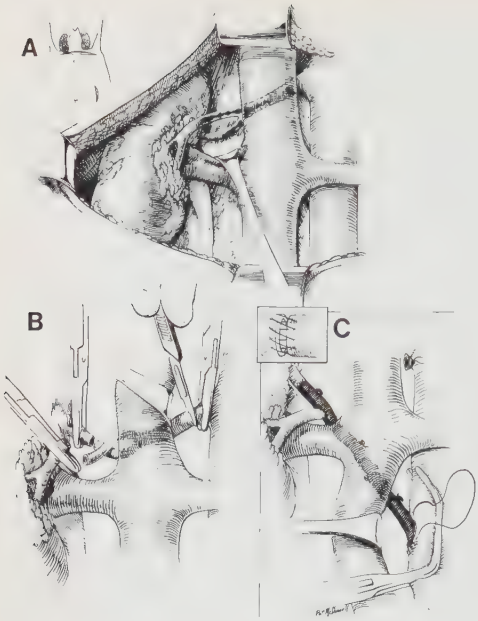


Figure 3. A method for revascularizing multiple segmental renal artery disease is illustrated.

distal anastomosis is completed. A large occluding clamp on the saphenous vein is detrimental and will cause the formation of scar tissue. Our only strictures within the vein graft have occurred when a large occluding clamp has been applied to the vein graft itself.

Many authors have emphasized, and we wholeheartedly agree, that meticulous dissection of the structures involved is paramount for the successful and easy angioplastic procedure. We have preferred to use a long upper abdominal transverse incision and mobilize either the left or right colon, depending upon the side, to visualize the kidney and the aorta. There are several advantages of this approach. It allows for free access to the entire vasculature of the kidney out to the hilum. It allows ready access to the aorta on either side, and thus seems to us to have many advantages over the midline approach. The anastomoses are always easily in view and there is seldom any difficulty in fashioning even the most distal anastomosis.

Multiple areas of stenosis can be handled readily utilizing this technique, and it has not often been necessary to explant the kidney (Fig. 3). Belzer et al. have utilized explantation of the kidney routinely for multiple distal lesions.[2, 12] By and large, we feel that this approach is not always necessary and that the absolute indications for explantation of the kidney are secondary operations on the renal artery or in patients who have traumatic lesions of the renal artery, either dissections or partial disruptions which have become invaded by scar tissue.

If one chooses to use the internal iliac artery routinely in place of the autogenous saphenous vein, care must be taken since this artery is

prone to fibrodysplastic disease and atherosclerosis.[12] In the event of severe atherosclerosis eversion endarterectomy should be done. However, in patients with extensive fibrodysplastic disease, involvement of the internal iliac artery precludes its use as a suitable arterial conduit.

On occasion we have used a Dacron prosthesis to revascularize the renal artery in patients with atherosclerotic disease. These operations are relatively straightforward and the renal arteries usually are large with adequate length of artery available for the anastomosis. Since there always is production of scar tissue around the anastomosis between Dacron and artery, one must be careful to do a large spatulated anastomosis so that this scar tissue will not occlude the lumen of the bypass. Impra or expanded Teflon may be a suitable bypass material for large renal arteries. Like Dacron, it has the disadvantage of not being tailorable to small vessels as autogenous tissue is.

Wylie has developed and championed the technique of transaortic endarterectomy.[21] This technique is suitable for selected patients in whom the disease process is primarily in the renal arteries and there is no associated aortic medial degeneration as seen in aneurysmal disease. The sine qua non of this operative procedure is extensive exposure of the upper aorta and both renal arteries. We prefer to mobilize the spleen, stomach, pancreas, and entire left colon in order to expose this portion of the aorta. Wylie prefers the midline approach. The aorta may be clamped at the diaphragmatic hiatus and arteriotomy made from the left of the superior mesenteric artery, curving to the midline to a point approximately one centimeter below the renal artery ostia. The aorta and the ostia of the renal arteries are endarterectomized and the renal arteries are everted into the lumen of the aorta in order to ensure a clean break. Care must be taken that there are no distal intimal flaps, and after the aortotomy is repaired, arteriograms must be done on the table in order to insure adequate flow and a satisfactory result. The clamp time to accomplish this procedure seldom is longer than thirty minutes. In Wylie's hands this operative procedure has been eminently successful, and the author agrees that in selected patients with bilateral disease it is the procedure of choice.

Occasional patients with fibrodysplastic disease involving multiple arteries may be suitably treated by dilation (Fig. 4). This technique causes fracture of the media of the involved vessel and will cause the artery to be widely patent. We have employed this technique on a limited basis in patients we felt were not suitable for bypass. The follow-up of these patients now approaches thirteen years with continued satisfactory results. Great caution must be exercised in dilation of renal vessels as the arterial wall is extremely thin and the risk of causing irreparable damage to the vessel wall with a small dilator is great. It must be emphasized that if dilation is done, it is a *secondary* procedure and should not be utilized as the procedure of choice, but only when bypass, either in situ or by explantation, is not a feasible procedure.

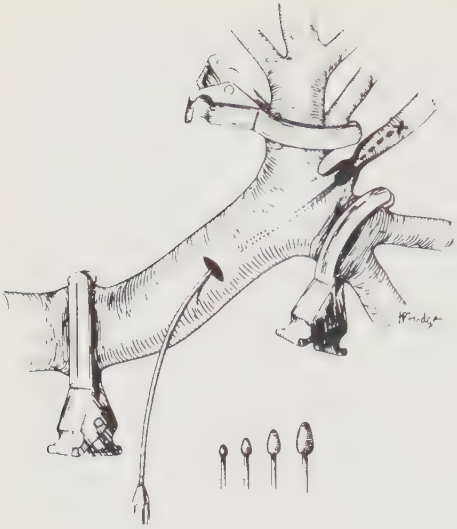


Figure 4. Technique used in dilation of renal artery lesions.

Results

The results of planned operative approach to renal artery stenosis are summarized in Table 1. It should be emphasized that the best opportunity for good results is in the younger population, and that the only mortality in the author's series was in the diffusely atherosclerotic individual.

Table 1. *Results of Planned Operative Approach to Renal Artery Stenosis*

	GROUP				
	1(N=27)*	2(N=132)†	3(N=54)‡	4(N=51)§	TOTAL
Postoperative status					
No. cured	24	76	17	13	130
No. improved	2	51	32	24	109
No. of failures	1	5	5	14‖	25
Response to surgical treatment, per cent					
Beneficial◐	96	96	91	73	94
Unfavorable	4	4	9	27	6

*Pediatric patients

†Young adults with fibrodysplasia

‡Focal renal arteriosclerotic disease

§Generalized arteriosclerotic disease

‖Three operative deaths in this category represent the only surgical mortality in all 254 patients

◐Includes cured and improved cases

REFERENCES

1. Baer, L., Parra-Carrillo, J. Z., Radichevich, I., et al.: Detection of renovascular hypertension with angiotensin II blockade. Ann. Intern. Med., *86*:257, 1977.
2. Belzer, F. O., Salvatierra, O., Palubinskas, A., et al.: Ex vivo renal artery reconstruction. Ann. Surg., *182*:456, 1976.
3. Bookstein, J. J., Walter, J. F., Stanley, J. C., et al.: Pharmacoangiographic manipulation of renal collateral blood flow. Circulation, *54*:328, 1976.
4. Ernst, C. B., Daugherty, M. E., and Kotchen, T. A.: Relationship between collateral development and renin in experimental renal artery stenosis. Surgery, *80*:252, 1976.
5. Fry, W. J., Brink, B. E., and Thompson, N. W.: New techniques in the treatment of extensive fibromuscular disease involving the renal arteries. Surgery, *68*:959, 1970.
6. Fry, W. J., Dean, R. H., Stanley, J. C., et al.: Symposium on renovascular hypertension. Contemp. Surg., *13*:49, 1978.
7. Fry, W. J., Ernst, C. B., Stanley, J. C., et al.: Renovascular hypertension in the pediatric patient. Arch. Surg., *107*:692, 1973.
8. Goldblatt, H., Lynch, J., and Hanzal, R. E.: Studies on experimental hypertensionI. The production of persistent elevation of systolic blood pressure by means of renal ischemia. J. Exp. Med., *59*:347, 1934.
9. Howard, J. W., and Connor, T. B.: Hypertension produced by unilateral renal disease. Arch. Intern. Med., *109*:8, 1962.
10. Hunt, J. C., and Strong, C. G.: Renovascular hypertension: mechanisms, natural history and treatment. *In* Laragh, J. H. (ed.): HypertensionManual: Mechanisms, Methods, Management. New York, Yorke Medical Books, 1974.
11. Marks, L. S., Maxwell, M. H., and Kaufman, J. J.: Renin, sodium and vasodepressor response to saralasin in renovascular and essential hypertension. Ann. Int. Med., *87*:176, 1977.
12. Olcott, C., and Wylie, E. J.: Renovascular hypertension. *In* Rob, C., (ed.): Advances in Surgery, Vol. 12. Chicago, Year Book Med. Publishers, 1978.
13. Palubinskas, A. J., and Ripley, H. R.: Fibromuscular hyperplasia in extrarenal arteries. Radiology, *82*:451, 1969.
14. Stamey, T. A., Nudelman, I. J., Good, P. H., et al.: Functional characteristics of renovascular hypertension. Medicine (Baltimore), *40*:347, 1961.
15. Stanley, J. C., Ernst, C. B., and Fry, W. J.: Fate of 100 aortorenal vein grafts: characteristics of late graft expansion, aneurysmal dilatation and stenosis. Surgery, *74*:931, 1973.
16. Stanley, J. C., and Fry, W. J.: Renovascular hypertension secondary to arterial fibrodysplasia in adults. Arch. Surg., *110*:922, 1975.
17. Stanley, J. C., and Fry, W. J.: Surgical treatment of renovascular hypertension. Arch. Surg., *112*:1291, 1977.
18. Stanley, J. C., Gewertz, B. L., and Fry, W. J.: Renal systemic renin indices and renal vein renin ratios as prognostic indicators in remedial renovascular hypertension. J. Surg. Res., *20*:149, 1976.
19. Stanley, J. C., Sottiurai, V., Fry, R. E., et al.: Comparative evaluation of vein graft preparation media: electron and light microscopic studies. J. Surg. Res., *18*:235, 1975.
20. Stoney, R. J., and Wylie, E. J.: Arterial autografts. Surgery, *67*:18, 1970.
21. Wylie, E. J.: Endarterectomy and autogenous arterial grafts in the surgical treatment of stenosing lesions of the renal artery. Urol. Clin. North Am., *2*:351, 1975.
22. Wylie, E. J.: Vascular replacement with arterial autografts. Surgery, *57*:14, 1965.
23. Wylie, E. J., Perloff, D., and Stoney, R. J.: Autogenous tissue revascularization technics in surgery for renovascular hypertension. Ann. Surg., *170*:416, 1969.

Department of Surgery
The University of Texas Health Science Center
5323 Harry Hines Boulevard
Dallas, Texas 75235

Aorto-Iliac Reconstruction

R. Clement Darling, M.D., David C. Brewster, M.D.,† John W. Hallett, Jr., M.D.,‡ and R. Clement Darling, III, B.S.§*

In the past two and one-half decades, substantial progress has been achieved in the surgical management of aorto-iliac occlusive disease. Advances in angiography and, more recently, the development of a variety of noninvasive testing methods have led to more accurate diagnosis and localization of the occlusive process. At the same time, improvements in materials for prosthetic grafts, surgical techniques and judgment, intraoperative anesthetic management, and postoperative care have generally led to excellent results with a steady reduction in operative mortality. Proper selection of patients and performance of an appropriate surgical procedure by an experienced surgeon are still the key determinants of success.

PATTERNS OF DISEASE

Segmental Aorto-iliac Disease (Type I and II)

The distal abdominal aorta below its renal branches is one of the most common sites of human arteriosclerosis. Appreciation of differing anatomic patterns of the occlusive process, as well as their natural history, is important in order to properly apply modern techniques for arterial reconstruction.

In our experience, in about one-third of patients with symptomatic aorto-iliac obliterative disease, the arteriosclerotic process is limited to the vessels above the inguinal ligament. Truly segmental aorto-iliac disease limited to the distal abdominal aorta and common iliac arteries (Type I) occurs in approximately 13 percent of patients (Figs. 1 and 2*A* and *B*). Slightly more than 20 per cent of patients have a Type

*Associate Clinical Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital, Boston, Massachusetts
†Assistant Professor of Surgery, Harvard Medical School; Assistant in Surgery, Massachusetts General Hospital, Boston, Massachusetts
‡Research Fellow, Harvard Medical School; Research and Clinical Fellow, Massachusetts General Hospital, Boston, Massachusetts
§Surgical Technician, Massachusetts General Hospital, Boston, Massachusetts

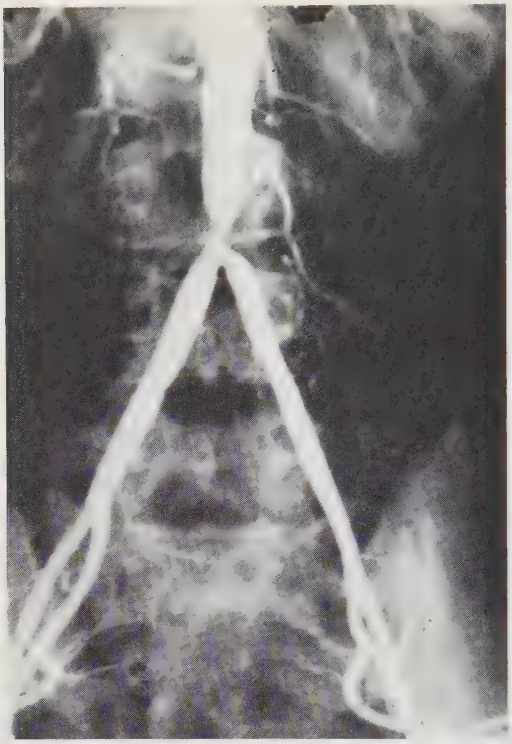


Figure 1. Segmental occlusion is limited to the distal aorta with extensive lumbar arterial collateral circulation.

II pattern denoting more widespread disease extending to the groin, that is, involving the external iliac or common femoral vessels in addition to the aortic bifurcation (Fig. 2, *C* and *D*).

Patients with segmental aorto-iliac disease (Type I) are characteristically younger, with a low incidence of hypertension and diabetes, but a high frequency of abnormalities of blood lipids. In contrast to the usual heavy male predominance (8:1 ratio) over females with arteriosclerotic obliterative disease in other locations, the distribution between the sexes with localized aorto-iliac disease is more uniform, particularly in patients below 50 years of age. One-half of the women will have had an artificial menopause produced either by radiation or hysterectomy. Essentially all such patients are cigarette smokers.[8]

Patients with localized aorto-iliac disease characteristically have symptoms classically described as the Leriche syndrome: absence of pulses in the lower limbs, proximal claudication of thigh, hip or buttock region, muscular atrophy of both lower limbs, and loss of potency in the male. Many patients will simply describe "weakness" of the legs when walking or may complain of gluteal tiredness when sitting or standing. An impressive number of such patients are studied because of chronic low back pain; often they have been seen initially by an orthopedist, neurologist, or neurosurgeon. A history of only proximal

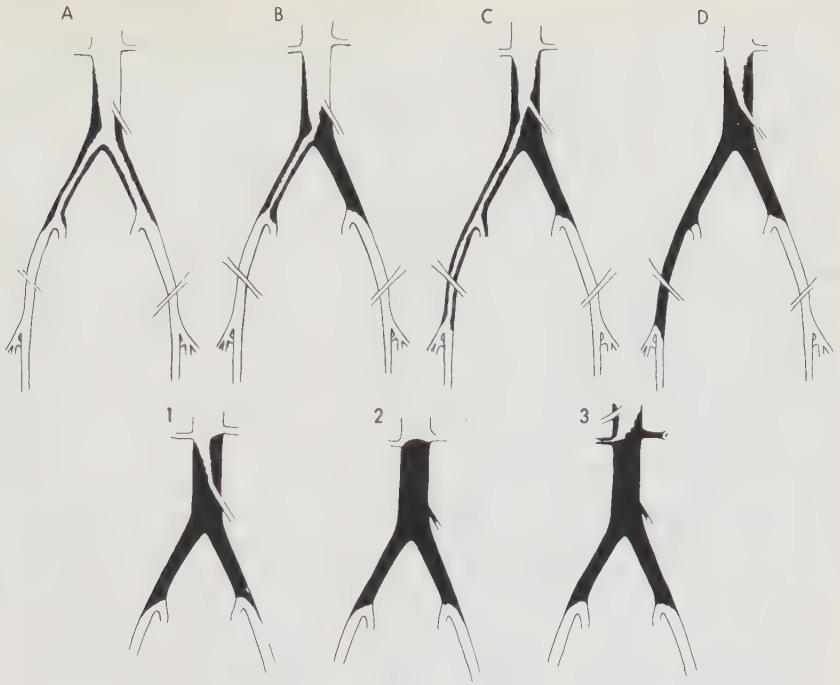


Figure 2. Anatomic patterns of aorto-iliac occlusive disease.

claudication almost always indicates the absence of significant occlusive disease below the inguinal ligament. On the other hand, about one-quarter of patients with isolated aorto-iliac occlusive disease will complain only of calf claudication. Although a history of impotence in a man frequently suggests involvement of the terminal aorta and common iliac arteries, this may have been overemphasized in the past and is now recognized to be closely related to the age of the patient, his general well-being, and other important psychosocial factors. No corresponding symptom in women has yet been recognized.

Patients usually have good nutrition of the skin of the lower limbs without ischemic ulceration, gangrene, or pain at rest. Changes in color and temperature are minimal. Although diminished, pulses in the lower limb may be palpable. Bruits over the iliac arteries or terminal aorta, which may be absent at rest, are usually easily discernible after exercise. Similarly, segmental pulse volume and Doppler pressure at the thigh, calf, and ankle will invariably be diminished after treadmill exercise and may provide valuable diagnostic information.[9, 16] The natural history of such localized aorto-iliac occlusive disease suggests that such patients have a more favorable prognosis, with less risk of the development of associated distal disease and therefore less chance of limb-threatening ischemia. Operation is usually based upon complaints of claudication, is almost always elective in nature, and should yield excellent results.

More commonly, the arteriosclerotic process progresses to involve the external iliac or femoral vessels (Fig. 2*C*), usually by a posterior tongue of atheroma. Although circulation to both lower extremities may be maintained via hypogastric collateral networks of the stenotic limb, the pattern of the disease is relatively unstable. Occlusion of the remaining common iliac artery may result in thrombus propagating to the groin, causing severe ischemia of both lower limbs (Fig. 2*D*) and precipitating the need for emergency arterial reconstruction, which is otherwise rarely necessary in chronic aorto-iliac obliterative disease.

Bilateral occlusion of the common iliac arteries usually results in distal aortic obstruction to the level of the inferior mesenteric artery (Fig. 2–1). If the vessel has undergone prior obliteration, superimposed thrombus extends proximally to just below the renal arteries which then act as the outflow tract for the proximal aorta (Fig. 2–2). Once in a while, one or both renal arteries may subsequently become occluded by progressive associated disease or by proximal extension of aortic thrombus with consequent anuria and acute renal failure (Fig. 2–3).

In most instances, the viability of the lower limbs is maintained by bridging collateral systems, bringing blood from the internal mammary, superior epigastric, and lower intercostal arteries, and producing reversed flow in the inferior epigastric and circumflex iliac arteries before realignment into the external iliac and femoro-profunda systems in the groin. Ill-advised lumbar sympathectomy in such instances may result in paradoxical development of gangrene in the lower limbs after division of these important collateral vessels. Perhaps more important in the patient with chronic aorto-iliac occlusive disease and absent femoral pulses is the visceral collateral arterial circulation which may develop. In some instances, the superior mesenteric artery may supply collateral flow to the limbs by means of a large collateral system involving the left colic artery. By anastomoses with the superior and inferior hemorrhoidal arteries and the hypogastric system, the left colic artery may provide the majority of collateral circulation to the lower limb (Fig. 3). Sigmoid resection or abdominoperineal operations in such patients may actually precipitate gangrene in the limbs.

Multilevel Occlusive Disease (Type III)

Unfortunately, the majority of patients (two thirds in our experience) have associated obliterative lesions of the arteries of the lower limb (Type III pattern). Such disease usually involves the femoropopliteal segment, the origin of the profunda femoris artery, or, less commonly, the tibial outflow system alone or in combination with the above. Patients are usually male (7:1) and older, and have a higher incidence of diabetes, hypertension, and associated cardiovascular disease elsewhere, particularly involving the coronary and cerebral vessels. Typically, the patient is a middle-aged or elderly man with a history of intermittent claudication of many years' duration, usually involving the calf muscles. Because of associated cardiac or other cardiovascular impairments, this is often overlooked by the patient and

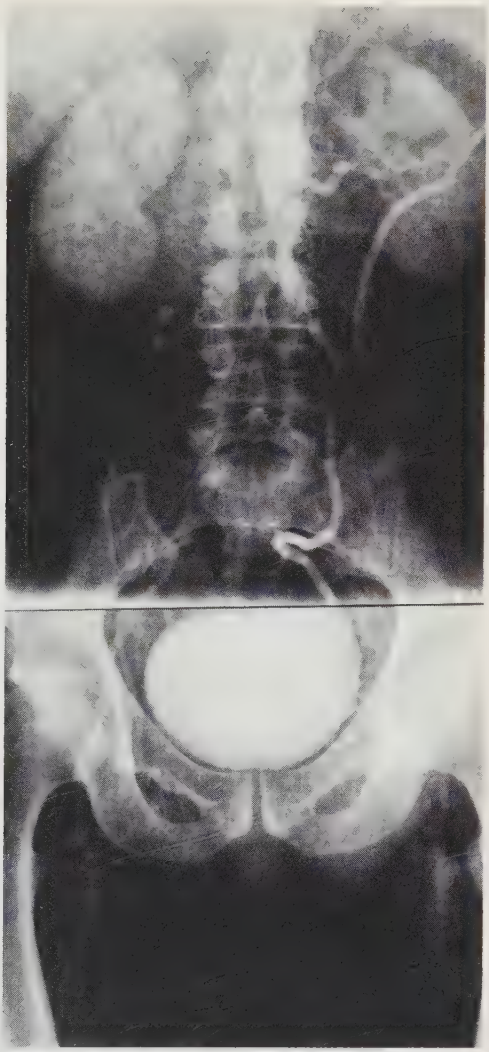


Figure 3. Lower limb viability in the presence of complete distal aorto-iliac occlusion may depend in part on a hypertrophied left colic artery.

his physician. However, as more proximal arteriosclerotic disease involving the aorto-iliac segment progresses, the femoral pulses weaken and intermittent claudication, which is still invariably limited to muscles below the knee, worsens. Disabling claudication, ischemic pain at rest, ulcer, or gangrene may develop. Examination of the lower limbs reveals poor nutrition of the skin, severe temperature changes, and pronounced rubor with dependency. The presence of ischemic lesions or gangrene almost always indicates such a combined segment (Type III) pattern of disease. The volume of the pulse and systolic pressures at the ankle are often barely perceptible at rest and disappear with exercise, which is often poorly tolerated. In contrast to patients with segmental aorto-iliac occlusion, maximum recovery time of determina-

tions of pulse volume before exercise, or systolic pressures at the ankle is remarkably prolonged.[9, 16]

Patients with combined segment disease, therefore, are much more prone to limb-threatening ischemia, and treatment is generally directed at maintaining limb survival. In most instances, proximal aorto-femoral reconstructions which are properly conceived and performed will remove the threat to limb viability, although complete relief of claudication may not be achieved by proximal operation alone.

TREATMENT

No medical treatment for aorto-iliac occlusive disease is effective once it has become symptomatic. Spontaneous improvement may occur in about 2 per cent of patients.[12] Medical treatment is directed almost entirely at impeding the atheromatous process by altering the risk factors in any given patient; that is, complete cessation of smoking, weight reduction, correction of lipid abnormalities, and treatment of hypertension. Although effective control of coexisting diabetes is desirable, this has not been proven to alter progression of the arteriosclerotic process. A properly planned exercise program to encourage the development of collateral circulation is important, as is good local foot care to avoid trauma and digital infection which so often are the precipitating causes of gangrene and amputation, particularly in patients with diabetes.

INDICATIONS FOR OPERATION

Indications for arterial reconstruction for occlusive disease have been generally well-defined and include disabling claudication, ischemic pain at rest, or ischemic skin lesions or frank gangrene. The latter two clearly imply threatened limb loss, and indicate the advisability of arterial reconstruction, if feasible, in all but the poorest risk patients. In general, age per se is not a major factor. In our experience, aorto-iliac reconstruction has been undertaken for limb-threatening ischemia in 52 per cent of patients.[3]

Some disagreement remains concerning the advisability of operation for claudication alone, and this decision must be clearly individualized. In general, claudication which impairs the occupational performance of the patient or significantly impairs the desired lifestyle of an otherwise good risk patient may be regarded as a reasonable surgical indication, providing a favorable anatomic situation for reconstruction exists. Such patients have usually been followed conservatively by the surgeon for a period of time, and the merits and possible risks of surgery have been thoroughly discussed. In general, we are more liberal in recommending surgery for claudication as a result of aorto-iliac disease than for that secondary to more distal disease because of the generally excellent and durable results achievable by aorto-iliac reconstruction.

Another less frequent but well-recognized indication for aorto-iliac reconstruction is the presence of peripheral atheromatous emboli resulting from proximal, usually ulcerated, aorto-iliac arteriosclerotic plaques. Clinical evidence of occlusive disease may not be apparent in such patients, and full peripheral pulses may be present without a history of claudication. Complete angiographic evaluation and recognition of this syndrome are mandatory.

Similarly, aorto-iliac disease may be associated with aneurysmal disease of the aorta. In our experience, approximately 15 per cent of patients undergoing elective resection of aortic aneurysms have associated arteriosclerotic narrowing of the iliac vessels. Occasionally, kinking of the iliac vessels by progressive dilatation and lengthening of the abdominal aorta produces a functional obstruction of one or both common iliac arteries. In some instances, the first sign of an aortic aneurysm may be clinical manifestations of chronic aorto-iliac occlusive disease.

Good preoperative angiography is generally accepted as a prerequisite prior to modern aortic reconstruction. Biplane studies, which provide oblique or lateral views, are highly desirable. Angiography is undertaken, however, only when a decision to operate has been reached on the basis of the above criteria.

While a decision for surgery is usually based upon the traditional clinical criteria and angiographic findings, the vascular laboratory may provide useful information. Noninvasive testing may help clarify the true severity of disease in the patient who is a poor historian, and may be particularly helpful in demonstrating progression of disease in the patient being followed on a conservative program.[16]

OPERATION

Controversy still exists regarding the optimal technique of aorto-iliac reconstruction, with proponents of endarterectomy and bypass grafting each claiming advantages (Fig. 4). More recently, some authors have proposed that extraanatomic reconstructions may be equally effective.

At present, the proper choice of operation depends on the general condition of the patient, the extent of the arteriosclerotic process, and the experience of the surgeon.

A recent analysis of the personal experience of one of the authors with aorto-iliac reconstruction over a 15 year period in 582 patients (1105 limbs) has demonstrated some significant trends, which together reflect our current methods and policies.[3] Separate analysis of approximately equal groups of patients operated on from 1963 to 1969 (Interval I) was performed and compared with those operated on from 1970 to 1977 (Interval II). While endarterectomy was performed in 72 per cent of patients in the earlier interval, graft procedures were selected in 89 per cent of patients in the later time period. Operative mortality was 5.1 per cent and early failure occurred in 4 per cent of patients in Interval I. In contrast, in Interval II, during which exten-

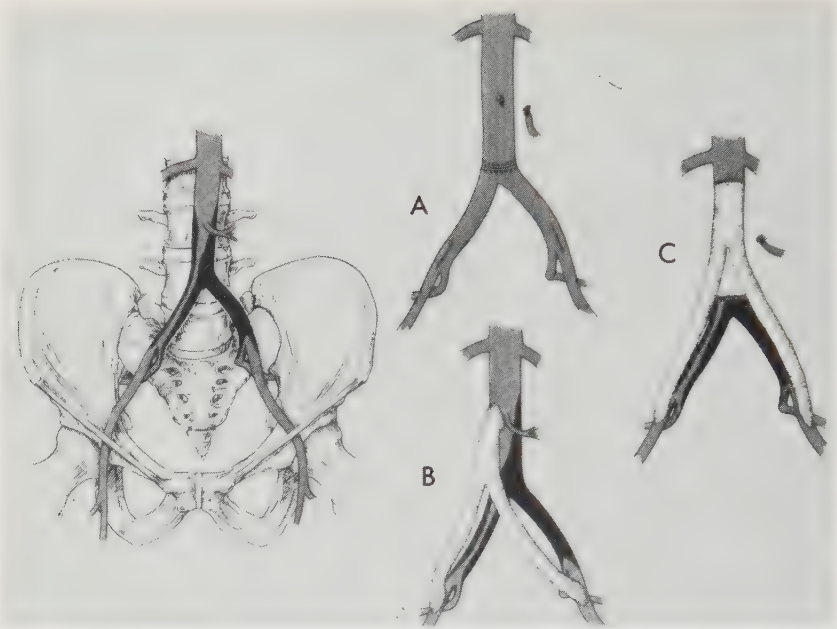


Figure 4. Aorto-iliac reconstruction commonly is carried out by endarterectomy (*A*) or aorto-iliac or aorto-femoral bypass with (*B*) or without (*C*) aortic resection.

sive endarterectomy procedures were essentially abandoned, operative mortality fell to only 2 per cent and early failure occurred in less than 1 per cent of patients. Long-term cumulative patency rates for the alternate procedures is indicated in Figure 5.

This experience suggests that in patients with localized aorto-iliac disease, either aorto-iliac endarterectomy or grafting yields excellent

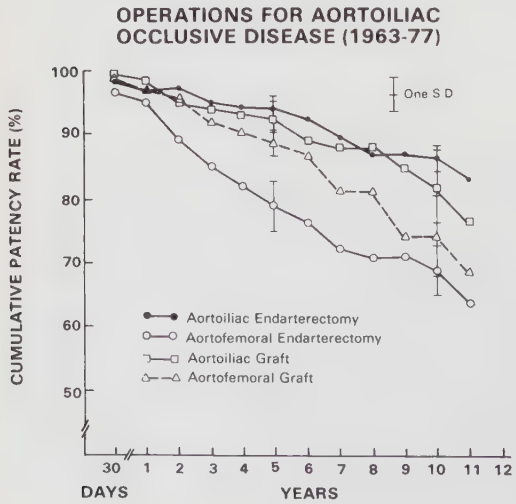


Figure 5. Cumulative patency rates in one of the author's personal experiences (R. Clement Darling, M.D.) with 582 patients undergoing aortoiliac reconstruction (1105 limbs). (*From* Brewster, D. C., and Darling, R. C.: Optimal methods of aortoiliac reconstruction. Surgery, *84*:739, 1978, with permission.)

early and late results. In such patients, there seems to be little difference between methods and much will depend upon experience of the surgeon with endarterectomy which is generally acknowledged to be a more demanding procedure.[11]

In our experience, endarterectomy will be suitable in approximately 10 per cent of patients with truly localized disease and is preferred because of the advantages of autogenous tissue and the absence of a prosthesis. Proper selection of patients for endarterectomy is of particular importance. Disease should terminate at or just beyond the common iliac bifurcation, allowing a satisfactory end point without extending more than 1 or 2 cm into the external iliac segment. This judgment is based on good preoperative angiography, often including oblique views of the pelvis, and is confirmed at the time of operation by palpation of the external iliac vessels. Complete disobliteration to the level of the external elastic lamina is nearly always possible despite extensive calcification in the arterial wall.

As shown in Figure 6, we believe endarterectomy is contraindicated in three instances. First, any evidence of aneurysmal change makes endarterectomy ill-advised since continued aneurysmal degeneration of the aortic adventitia may be expected. Second, bypass grafts are preferable if the aorta is occluded to the level of the renal vessels. In this situation, endarterectomy may require considerably more expo-

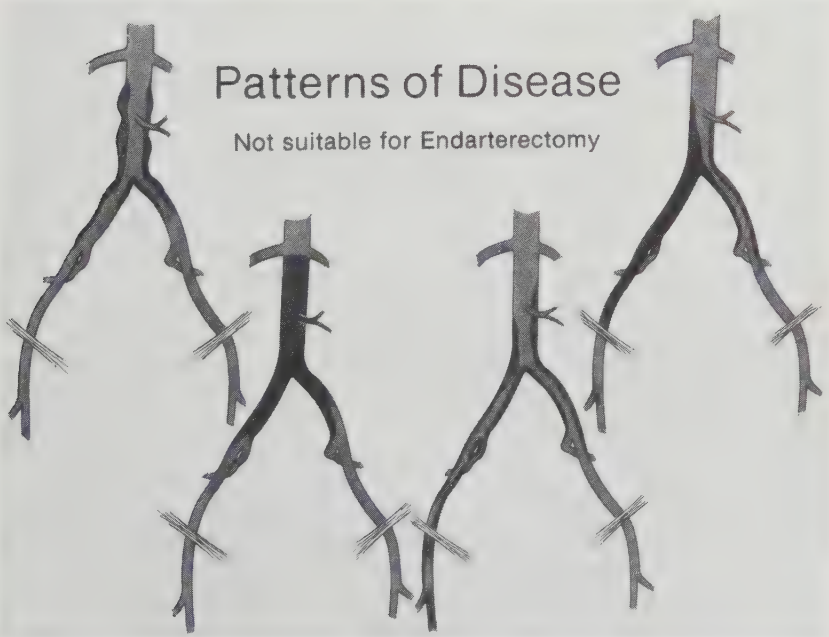


Figure 6. In the authors' experience, endarterectomy, though the most durable of aorto-iliac reconstructions, is contraindicated if there is (left to right): aneurysmal degeneration, aortic occlusion to the renal artery level, or if the external iliac or femoral arteries are involved. In these situations aortic resection and bypass are preferable.

sure and loss of blood. Simple transection of the aorta distal to the renal arteries with thrombectomy of the aortic cuff and insertion of an aortic prosthesis may be more safely and expeditiously carried out. Finally, significant extension of the disease process to the external iliac segment will make bypass grafting preferable in the majority of patients. Difficulties with adequate endarterectomy of the external iliac artery as a result of its small size, greater length, somewhat more difficult exposure, and the more muscular and adherent nature of its medial layer are well recognized. Such considerations led to a higher incidence of early occlusion and late failures due to recurrent stenosis in late follow-up studies.[7, 3]

For these reasons, we believe more extensive disease is best managed by bypass grafting, which has demonstrated a lower mortality rate, a lower initial failure rate, and better late patency. Although distal anastomosis may be done at the distal external iliac level, this is frequently more difficult, and more importantly, is associated with a higher late failure rate as a result of progressive disease beyond the anastomosis.[1, 3, 7] Aorto-femoral bypass grafts, therefore, appear to currently represent the procedure of choice in the majority of patients. Fears that a great incidence of infection or false aneurysm would occur if grafts were extended to the femoral level have not been documented.[13, 14] Our own experience indicates an incidence of infection of only 0.3 per cent and the formation of false aneurysms in 1.4 per cent of patients undergoing aorto-femoral grafts.[3] We believe the incidence of infection may be kept to an absolute minimum by meticulous sterile technique, the use of adhesive drapes to limit the exposure of the skin, and the use of a broad-spectrum antibiotic begun preoperatively and continued for three to five days postoperatively. Similarly, the incidence of false aneurysm should be small if femoral anastomosis is done without tension to a segment of common femoral artery which has not been endarterectomized. Similarly, we believe the use of permanent suture material such as braided polyester is preferable to the use of monofilament such as polyethylene and polypropylene. Although the latter material may prove satisfactory, long-term studies are not yet available to indicate the absence of late fracture or other problems which might lead to a higher incidence of false aneurysm formation.

During a review of our experience with aorto-femoral grafts, it became apparent that a significant difference in late patency rates existed between grafts done in intervals I and II (Fig. 7). In our opinion, this is attributable to a number of factors. First, more reliable prosthetic grafts are now available. Our own preference is for a high-quality knitted Dacron prosthesis, such as that manufactured by Golaski Laboratories and used in the majority of patients in interval II.

Second, analysis of limb failures revealed that most were associated with a Type I femoral anastomosis (Fig. 8). While this may be satisfactory in the first few years, particularly in patients with an open distal system, the development of any progressive occlusive disease of the superficial femoral artery or in the origin of the profunda femoris artery favors reduced flow and eventual limb occlusion. For this reason, in

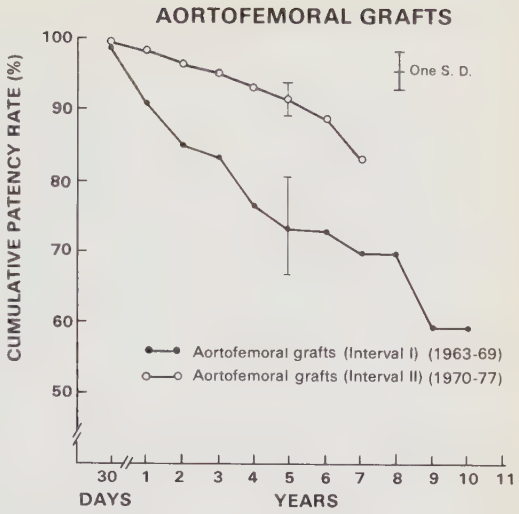


Figure 7. A comparison of cumulative patency rates of aorto-femoral grafts according to interval. (*From* Brewster, D. C., and Darling, R. C.: Optimal methods of aortoiliac reconstruction. Surgery, 84:739, 1978, with permission.)

Interval II the tip of the beveled graft hood was carried across the origin of the profunda as a patch angioplasty (Type III or IV, Fig. 8) whenever significant disease was present in the superficial femoral system or origin of the profunda femoris artery. We feel that it is also important to use 3 to 5 fine 5–0 or 6–0 interrupted permanent sutures at the distal apex of the anastomosis of the profunda femoris artery to avoid possible compromise of this outflow tract. We believe preservation of an adequate outflow tract in the profunda has been a significant factor in the superior late patency of the most recent time period (91 per cent cumulative limb patency at 5 years) (see Fig. 7).

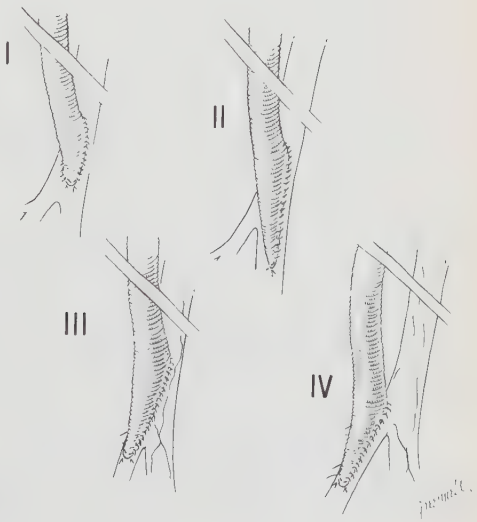


Figure 8. Four methods of distal reconstruction are shown. Types III and IV are favored because of the high incidence of preexisting or subsequent stenosis of the profunda artery.

Figure 9. The so-called "onlay" bypass (*A*) has fallen into disfavor because of the high incidence of late failure caused by recurrent aortic disease (*B*). It may require secondary aortic resection and bypass (*C*).

In our experience, end-to-end proximal anastomoses are preferable to end-to-side or onlay techniques. The end-to-side technique was associated with an incidence of limb failure at 5 years of 44 per cent in Interval I as opposed to a failure rate of only 15 per cent during the same time period with end-to-end anastomosis. Although an end-to-side technique theoretically saves collateral vessels, intraaortic thrombus may be dislodged and carried to the lower extremities when tangential clamps are applied to the diseased aorta. Since the host aorta commonly occludes later, end-to-side anastomosis is hemodynamically less satisfactory. Furthermore, such an anastomosis is often, by necessity, placed low on the aorta at the site of significant atheromatous disease which may well progress with time (Fig. 9). Finally, resection of a small section of aorta and insertion of a graft in end-to-end fashion allows easier and more complete tissue coverage. Most aortoduodenal fistulas reported have occurred following end-to-side anastomosis and may have resulted from the recognized difficulty of reperitonealization and separation of graft from bowel in this type of aortic bypass.

End-to-side anastomosis may be considered if significant accessory renal arteries arise from the diseased aorta, or if the inferior mesenteric artery is unusually large with poor back bleeding suggesting poor collateral flow, particularly if the left colon appears to be compromised. However, in most such instances we prefer reimplantation of such vessels into the aortic prosthesis rather than onlay bifurcation grafting.

The role of concomitant lumbar sympathectomy at the time of aortic reconstruction remains unsettled. Since it is generally accepted that sympathectomy does increase total blood flow to the limbs and

skin, it is our policy to carry out limited (L2 to 3) sympathectomy in conjunction with aortic operation whenever occlusive disease is present below the inguinal ligament. This is easily and quickly accomplished.

No definite conclusions seem possible in regard to the incidence of postoperative sexual dysfunction associated with alternate methods in the male patient. With care to preserve autonomic nerve fibers as much as possible by minimizing dissection in the region of the aortic bifurcation, erectile function can be preserved in most patients if present preoperatively;[10] retrograde ejaculation may occur more frequently, but is less troublesome to most patients.

Intraoperative monitoring using recordings of pulse volume at the calf or ankle has all but eliminated early graft failure. Early postoperative occlusions are usually the result of technical errors, such as thromboemboli from the upstream aorta or clot improperly flushed from the prosthetic limb prior to restitution of flow, twisting or kinking of the limb in the retroperitoneal tunnel, or elevation of a downstream arteriosclerotic plaque or flap. Such problems may almost always be recognized on the table with the use of such intraoperative monitoring, occasionally supplemented by intraoperative arteriography.

CONTINUING PROBLEMS

A continuing important and vexing problem is whether to extend aorto-femoral grafting to the popliteal level at the time of initial operation. Previous reviews have suggested that approximately 25 per cent of patients with combined segment disease will fail to achieve relief of ischemic symptoms following proximal reconstruction alone.[15, 18, 19] Although most surgeons have abandoned simultaneous aorto-femoropopliteal procedures, the issue remains largely unanswered. In our experience, technically successful proximal operation will satisfactorily relieve symptoms of claudication or ischemic pain at rest, and simultaneous reconstruction to the femoropopliteal level is best avoided. However, a small percentage of patients with severe ischemic lesions or impending gangrene may require simultaneous multilevel reconstruction; their selection remains difficult.[1, 6] Several guides are available, the first of which is the clinical setting. A patient with a diminished but present femoral pulse and who has a severe open ischemic foot lesion and distal disease, may require a combined aorto-femoral and downstream procedure to prevent loss of a limb. The relative hemodynamic significance of the aorto-iliac component of combined multileveled disease may be best defined by measurements of femoral artery pressure at the time of preoperative angiography.[2, 4] An aorto-femoral pressure gradient at rest of greater than 5 mm of mercury or a fall of femoral artery pressure greater than 15 per cent with reactive hyperemia indicates hemodynamically significant proximal disease and the likelihood that an upstream procedure alone will suffice. On the other hand, a negative femoral artery pressure suggests

that it is unlikely that the patient will be satisfactorily relieved of ischemic symptoms, and a more distal reconstruction must be considered.

Several other preoperative features may suggest the advisability of simultaneous grafting for severe ischemia. Angiography may suggest a poorly developed or diffusely diseased profunda femoris system. Similarly, angiographic demonstration of popliteal occlusion below the usual level of collateralization of the profunda femoris artery with the geniculate system may suggest a poor response to proximal reconstruction alone. Frequently such patterns of disease are suggested by the findings in the vascular laboratory of a resting ankle pressure less than 30 mm of mercury, absent resting Doppler signals at the ankle, or a flat recording of pulse volume at rest.

If preoperative vascular studies reveal diminished segmental pressures but show that the pressure gradient from thigh to ankle is 20 mm of mercury or less, our experience has suggested that the profunda system is probably well developed and that distal reconstruction will probably not be necessary. Further investigation is continuing, to evaluate the usefulness of such noninvasive criteria.

Finally, several intraoperative criteria may be utilized. Restoration of an improved pulse volume recording at the calf or ankle, as compared to preoperative tracings, can give reassurance of satisfactory improvement in distal circulation and obviate an associated distal reconstruction. However, improvement of pulse volume recordings or Doppler ankle pressures may not be immediately apparent in the presence of significant distal disease, especially in the cold vasoconstricted patient. A very useful intraoperative guide in predicting a good clinical response may be assessment of the anatomic size of the profunda femoris vessel itself. If the proximal profunda artery accepts a 4 mm probe and if a No. 3 Fogarty embolectomy catheter can be passed for a distance of 20 cm or more, it is likely that the arterial system of the profunda femoris artery is well developed and will function as a satisfactory outflow tract.[5]

SUMMARY

Chronic aorto-iliac occlusive disease is commonly manifested by progressive intermittent claudication limited to the buttock and thighs when the disease is segmental, and by calf claudication or ischemic pain, ulceration, or gangrene when it coexists with occlusive disease in the femoro-popliteal or tibial vessels. No medical treatment is effective once aorto-iliac occlusive disease has developed. In properly selected patients, the optimal treatment is aorto-iliac reconstruction either by means of endarterectomy if the occlusive process is localized or by insertion of an aorto-femoral Dacron bypass graft if obliterative disease is extensive. Since most patients initially have intermittent claudication only and are not in danger of losing a limb, any operative procedure should be considered elective and should be done by an experienced surgeon with anticipation of a low operative mortality and

good long-term function. Complete cessation of cigarette smoking,[17] maintenance of lean weight, and daily exercise programs are considered important in lessening the progression of the basic atherosclerotic disease in the reconstructed arterial segments as well as in other vessels.

ACKNOWLEDGMENT

The authors express their appreciation to Miss Karin Kutrieb and Miss Geri Flynn for editing the manuscript.

REFERENCES

1. Baird, R. J., Feldman, P., Miles, J. T., et al.: Subsequent downstream repair after aorta-iliac and aorta-femoral bypass operations. Surgery, *82*:785, 1977.
2. Brener, B. J., Raines, J. K., Darling, R. C., et al.: Measurement of systolic femoral arterial pressure during reactive hyperemia: an estimate of aorto-iliac disease. Circulation, *49* and *50* (suppl. II):259, 1974.
3. Brewster, D. C., and Darling, R. C.: Optimal methods of aortoiliac reconstruction. Surgery, *84*:739, 1978.
4. Brewster, D. C., Waltman, A. C., O'Hara, P. J., et al.: Femoral artery pressure measurement during aortography. Circulation, in press.
5. Buckley, C. J.: Personal communication.
6. Collins, G. J., Rich, N. M., Andersen, C. A., et al.: Staged aortofemoropopliteal revascularization. Arch. Surg., *113*:149, 1978.
7. Crawford, E. S., Manning, L. G., and Kelly, T. F.: "Redo" surgery after operations for aneurysm and occlusion of the abdominal aorta. Surgery, *81*:41, 1977.
8. Darling, R. C.: Medical progress: peripheral arterial surgery. New Engl. J. Med., *280*:26, 84, 141, 1969.
9. Darling, R. C., Raines, J. K., Brener, B. J., et al.: Quantitative segmental pulse volume recorder: a clinical tool. Surgery, *72*:873, 1972.
10. DePalma, R. G., Levine, S. B., and Feldman, S.: Preservation of erectile function after aortoiliac reconstruction. Arch. Surg., *113*:958, 1978.
11. Gaspard, D. J., Cohen, J. L., and Gaspar, M. R.: Aortoiliofemoral thromboendarterectomy vs. bypass graft. Arch. Surg., *105*:898, 1972.
12. Humphries, A. W., deWolfe, V. G., Young, J. R., et al.: Evaluation of the natural history and the results of treatment in occlusive arteriosclerosis involving the lower extremities in 1,850 patients. *In* Wesolowski, S. A., and Dennis, C. (eds.): Fundamentals of Vascular Grafting. New York, McGraw-Hill, 1963, pp. 421–439.
13. Malone, J. M., Moore, W. S., and Goldstone, J.: The natural history of bilateral aortofemoral bypass grafts for ischemia of the lower extremities. Arch. Surg., *110*:300, 1975.
14. Moore, W. S., Cafferata, H. T., Hall, A. D., et al.: In defense of grafts across the inguinal ligament: an evaluation of early and late results of aorto-femoral bypass grafts. Ann. Surg., *168*:207, 1968.
15. Perdue, G. D., Long, W. D., and Smith, R. B.: Perspective concerning aorto-femoral arterial reconstruction. Ann. Surg., *173*:940, 1971.
16. Raines, J. K., Darling, R. C., Buth, J., et al.: Vascular laboratory criteria for the management of peripheral vascular disease of the lower extremities. Surgery, *79*:21, 1976.
17. Robicsek, F., Daughterty, H. K., Mullen, D. C., et al.: The effect of continued cigarette smoking on the patency of synthetic vascular grafts in Leriche syndrome. J. Thorac. Cardiovasc. Surg., *70*:107, 1975.
18. Royster, T. S., Lynn, R., and Mulcare, R. J.: Combined aortoiliac and femoropopliteal occlusive disease. Surg. Gynecol. Obstet., *143*:949, 1976.
19. Sumner, D. S., and Strandness, D. E.: Aortoiliac reconstruction in patients with combined iliac and superficial femoral arterial occlusion. Surgery, *84*:348, 1978.

Massachusetts General Hospital
Boston, Massachusetts 02114

Femoro-Popliteal and Femoro-Tibial Reconstructions

*John A. Mannick, M.D.**

Femoro-popliteal arterial reconstruction has been one of the most widely applied techniques for revascularization of the ischemic lower extremity since the early 1950s. However, the procedure remains controversial, both with respect to its indications and its long-term effectiveness.[3, 10, 22, 23, 26, 33] Femoro-tibial reconstructions have been performed in sizable numbers for about 15 years, but are even more controversial. Many surgeons avoid tibial vessel grafts entirely because of expectation of poor results.[25, 31, 36]

INDICATIONS FOR SURGERY

Salvage of a limb is the most obvious indication for femoro-popliteal and femoro-tibial reconstruction. Significant arteriosclerotic disease of the femoro-popliteal and/or tibial systems is the most common cause of threatened limb loss in our hospital. The population of patients in danger of amputation from severe femoro-popliteal-tibial occlusive disease is, in general, elderly with an average age in the late 60s or early 70s. In our experience about a third are diabetic; the diabetic group is likely to be at the younger end of the age spectrum. These patients present with obvious signs of ischemia at rest and almost invariably have chronic pain in the foot when at rest. In addition, they may have nonhealing ischemic ulceration of the foot or ankle, pregangrenous changes of the skin, or frank gangrene. Gangrene itself does not rule out successful limb salvage provided it involves a limited enough area to permit successful ambulation on the foot when the gangrenous tissue is ultimately excised. Patients who present with extensive gangrene of the foot or lower leg clearly require primary amputation rather than arterial reconstruction.

A minority of patients with advancing femoro-popliteal occlusive disease may present with a sudden onset of ischemic pain in the foot

*Moseley Professor of Surgery, Harvard Medical School, Boston; Surgeon-in-Chief, Peter Bent Brigham Hospital, Boston, Massachusetts

at rest and a history compatible with sudden occlusion of the superficial femoral artery. These individuals are not necessarily candidates for limb salvage operations because in many instances conservative management in bed with anticoagulants and the reverse Trendelenberg position will permit, in a few days' time, a significant increase in perfusion of the distal extremity by means of an enlarging collateral circulation. Obviously, careful observation of these individuals is mandatory and conservative management should be abandoned promptly if progressive improvement in the circulation to the distal extremity fails to occur. However, a significant percentage of individuals will recover from such an event without the necessity of arterial surgery for limb salvage.

On clinical examination the patient who presents with compromised viability of a limb from chronic femoro-popliteal-tibial occlusive disease will have a femoral pulse in the affected extremity of moderately good to normal quality and there will be no palpable pulses below that level. Pressure measurements using the Doppler instrument will ordinarily reveal an ankle pressure of 60 mm of mercury or less, and an ankle to systemic pressure index of 0.4 or below. We have not found more elaborate methods of noninvasive evaluation of these patients' circulatory status to be of value.

Claudication is the second major indication for femoro-popliteal reconstructive surgery. We do not consider it appropriate to perform femoro-tibial reconstruction for symptoms of claudication because of the uncertain outcome of this method of therapy.[16, 23, 33] It is clear from a number of studies of the natural history of claudication caused by femoro-popliteal occlusive disease that the patient with claudication has a good prognosis with respect to limb viability over the first five years following the onset of his symptoms without surgery of any kind.[15, 27] Thus the degree of disability caused by the claudication must be the determining factor in selecting such patients for femoro-popliteal arterial surgery. The most obvious example of disability is, of course, the patient who is unable to work because of his or her claudication, a factor greatly dependent upon the nature of the patient's occupation. Claudication that is disabling for a longshoreman or construction worker may be only a minor nuisance to an office worker. Patients presenting with claudication as the primary complaint with femoro-popliteal occlusive disease ordinarily are a decade younger than those in danger of the loss of a limb. These patients ordinarily will not have evidence or symptoms of ischemia at rest and will usually have warm, well-perfused feet on examination in the office. Ankle pressures measured with the Doppler instrument will almost invariably be greater than 60 mm of mercury and are sometimes in the 100 mm of mercury range at rest. A considerable lowering of ankle pressure, however, will be produced by modest exercise of the affected extremity.

There is a group of patients presenting with the primary complaint of rather severe claudication coming on at short distance, usually less than a block of level walking at a modest pace, who have hemody-

namic evidence of ischemia at rest even though they have no pain. Doppler ankle pressures at rest, in this group of patients, will be 60 mm of mercury or below and the ankle to systemic pressure index will be below 0.4. We have considered such objective signs of severe occlusive disease to be an added indication for surgery in such patients but have not chosen to classify these individuals as limb salvage patients when considering the late results of reconstructive surgery.

While physical examination and the use of simple noninvasive studies, such as measurement of sequential limb pressures with the Doppler instrument, are helpful in evaluating the symptomatic patient and making a decision for or against surgical intervention, complete angiography is essential for the adequate planning of an operation for arterial reconstruction. We prefer to get a complete picture of the arterial supply to the lower extremities in each instance by the retrograde Seldinger catheter technique. This includes views of the abdominal aorta, iliac arteries, the femoral bifurcations, the complete course of the superficial femoral and popliteal arteries and the tibial vessels in the lower leg, ankle, and foot. We also routinely request oblique views of the pelvic vessels and groins in order to be confident of ruling out hemodynamically significant lesions of the terminal aorta or iliac arteries which would require more proximal arterial reconstruction and to look for the presence of lesions at the origin of the profunda femoris arteries often not seen in conventional anteroposterior views.

OPERATIVE PROCEDURES

The preferred material for arterial replacement in the femoropopliteal-tibial system is the reversed autogenous saphenous vein. We believe that it is particularly important to use a bypass graft of autogenous vein in patients being operated upon for limb salvage because these individuals are much more likely to have extensive occlusive disease with narrowing of portions of the popliteal artery and one or more of the tibial vessels as well as occlusion of the superficial femoral artery. Therefore, the reconstruction will ordinarily be required to stay open initially under conditions of restricted outflow and low mean graft flow rates sometimes in the order of 50 ml per min or less.[24, 28] The saphenous vein appears to be well suited for these conditions since it is autogenous tissue and, therefore, is not subject to the host transplantation immune response or foreign body reaction. It also remains supple through the years and can cross joint lines without kinking itself or the host artery. Finally, the vein graft, if handled properly, possesses a viable clot-resistant intimal lining. There is no other material available with all of these desirable characteristics except autogenous artery, which is ordinarily impossible to obtain in sufficient length to use for a graft in patients with far-advanced femoro-popliteal occlusive disease.

However, in order to be suitable for use the vein graft must be of satisfactory size and quality. The vein must be free of phlebosclerosis

and must contain no areas of fibrous thickening that result from old phlebitis and recanalization. In addition, the vein graft should be of adequate size, probably at least 4 mm in external diameter when gently dilated. This will permit the inevitable hypertrophy of the vein intima, which occurs upon subjecting the vein graft to arterial pressure, to take place without narrowing the lumen to a degree which seriously interferes with blood flow. If a suitable saphenous vein is not present we have not hesitated to use an arm vein, either cephalic or basilic, as a substitute.[5, 18] Again the same strict criteria must be used for accepting these veins as arterial substitutes.

If no suitable vein is available our second choice is currently the expanded polytetrafluoroethylene (PTFE) graft which, in the short-term follow-up reports currently available[3, 12, 37, 38] and in our own limited experience, has functioned better than other prosthetic materials in the femoro-popliteal system. However, in our hands these grafts have not functioned as well as autogenous vein grafts in patients with far-advanced femoro-popliteal-tibial occlusive disease.

In patients operated upon for claudication the choice of graft materials may be broader, particularly if the arterial tree from the mid-popliteal level distally is relatively normal. This permits the lower anastomosis of a femoro-popliteal graft to be placed above the level of the knee joint. In such patients 6 or 8 mm grafts of knitted Dacron have often functioned well for many years. However, even in individuals with claudication on the basis of superficial femoral artery occlusion and excellent run-off we prefer to use autogenous vein grafts whenever possible.

In certain patients with very localized superficial femoral artery obstructions at the level of the adductor canal we have utilized local endarterectomy and vein patch grafting as a simple substitute for femoro-popliteal bypass vein grafting. Reports from others[17] and our own limited experience suggest that this procedure in selected individuals has excellent long-term success.

Femoro-Popliteal Reconstruction

In patients operated upon for limb salvage it is most often necessary to place the distal anastomosis of the vein graft in the distal popliteal artery since this segment is ordinarily the last to be involved by arteriosclerosis. This segment of the artery is best approached through an incision placed on the medial aspect of the leg extending from the level of the knee joint distally over the course of the greater saphenous vein which will be harvested through the same wound (Fig. 1). After the fascia is divided, the medial head of the gastrocnemius can be retracted proximally and posteriorly, exposing the distal popliteal artery and its companion structures.

We ordinarily attempt to place the proximal anastomosis of the vein graft in the common femoral artery because this vessel again is likely to remain free of hemodynamically significant occlusive disease in patients with far-advanced arteriosclerosis. We use a longitudinal groin incision placed directly over the femoral vessels. We have aban-

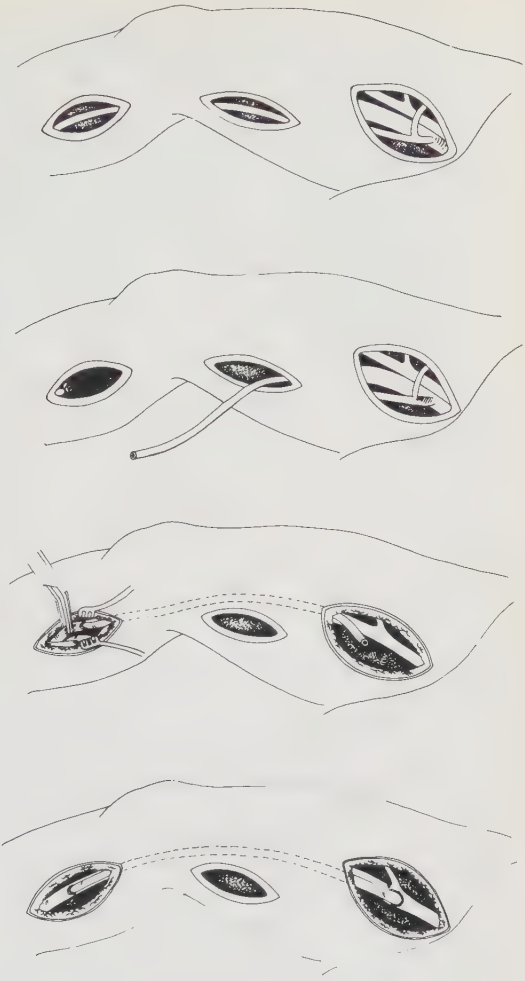


Figure 1. For grafting of the femoral-popliteal saphenous vein, the saphenous vein is exposed through a series of incisions. The upper and lower incisions are also used to expose the common femoral artery and the distal popliteal arteries respectively. After removal and gentle dilatation of the saphenous vein, it is first reversed and sutured end-to-side to the distal popliteal artery, then drawn through a subsartorial tunnel into the proximal incision where it is sutured to the common femoral artery.

doned oblique and transverse groin incisions because of the tendency to divide lymphatic channels and because frequent lymph leaks and lymphoceles were encountered when these incisions were used. The saphenofemoral junction is easily located in the groin incision and the greater saphenous vein is traced distally through a series of smaller incisions placed on the medial aspect of the thigh and into the distal incision. We believe that healing is more easily accomplished with a series of smaller incisions than with one large incision over the entire course of the saphenous vein. It is important, however, to place the incision directly over the saphenous vein because undermining the skin during the course of dissecting out the vein frequently causes slough in elderly patients with peripheral vascular disease. All branches of the saphenous vein are ligated with fine silk and divided as they are encountered. Blood is allowed to flow through the vein

Figure 2. The saphenous vein has been removed and is being gently dilated with chilled heparinized Ringer's lactate solution.

until all branches have been interrupted. Then the vein is quickly divided proximally and distally and removed. The vein is immediately flushed with chilled heparinized Ringer's lactate solution and cooled to a temperature of 4° C. It is gently dilated with the same solution, with care to avoid damage to the vein wall, in order to assess the quality of the wall and the adequacy of the vein caliber, and to permit division of any constricting adventitial bands and ligation of any branches that were missed during the dissection (Fig. 2).

We ordinarily prefer to place the vein graft in anatomical position along the course of the artery it replaces and to make a tunnel by blunt dissection joining the proximal and distal incisions passing between the heads of the gastrocnemius and beneath the sartorius muscles.

Prior to performing any anastomoses systemic heparin is administered in a dose of approximately 100 units per kg. For femoro-popliteal bypass grafting the distal anastomosis is always performed first. A longitudinal arterotomy is made in the popliteal artery for a distance approximately twice the diameter of the graft to be used. The arterotomy is held open with stay sutures. For vein grafts the Kunlin[20] technique of anastomosis is used, in which double armed sutures are started at both ends of the anastomosis and projected toward the middle of each side (Fig. 3). The suture material is ordinary 6-0 or 5-0 polypropylene. For plastic grafts we use the standard technique, beginning a suture at the heel end of the bevelled graft and running one side well around the toe of the graft, thus permitting maximum visualization while placing sutures in the outflow of the graft, before closing the opposite side with the other end of the double armed suture. It is our practice to calibrate the outflow of the anastomosis with coronary artery dilators just prior to completion of the last few stitches.

It is very important not to injure the intima of the popliteal artery with vascular clamps. We prefer to use Fogarty clamps, equipped with silicone-filled pads on the jaws, which appear to be minimally trau-

Figure 3. The Kunlin technique of vascular anastomosis is used for femoral popliteal vein grafting.

matic. After completion of the distal anastomosis of a vein graft, the graft is again filled with chilled Ringer's lactate solution, and under perfect alignment drawn through the tunnel into the proximal incision utilizing a large clamp or commercial tunneling instrument. Ringer's solution is again flushed down the graft to ensure there are no rotary deformities in graft placement. The graft is then carefully held in perfect alignment with the forceps, trimmed, and split longitudinally for anastomosis with the common femoral artery, using the same technique as is employed in the popliteal anastomosis (Fig. 4). It is very important in performing the femoral anastomosis, which is usually made with the smallest portion of the reversed saphenous vein graft, to take delicate bites of the vein and avoid incorporating too much of the wall of the vein into the anastomosis so as not to narrow the inflow into the graft.

At the time of performing the femoral anastomosis the origin of the profunda femoris artery is routinely inspected and calibrated with coronary artery dilators. An orifice of 4 mm in diameter is considered to be acceptable. If the profunda femoris is shown to be constricted by angiography or by inspection at the time the proximal anastomosis is performed, the profunda stenosis is corrected by profundaplasty with or without accompanying endarterectomy.

While profundaplasty has received a good deal of publicity as an alternative to femoro-popliteal grafting for patients with femoro-popliteal occlusive disease,[2, 7, 14, 21, 26] we have found in our practice that only 10 per cent of individuals with predominantly femoro-popliteal-tibial occlusive disease have significant lesions of the profunda femoris artery. We have preferred to repair these profunda lesions concomitantly with femoro-popliteal or femoro-tibial grafting rather than

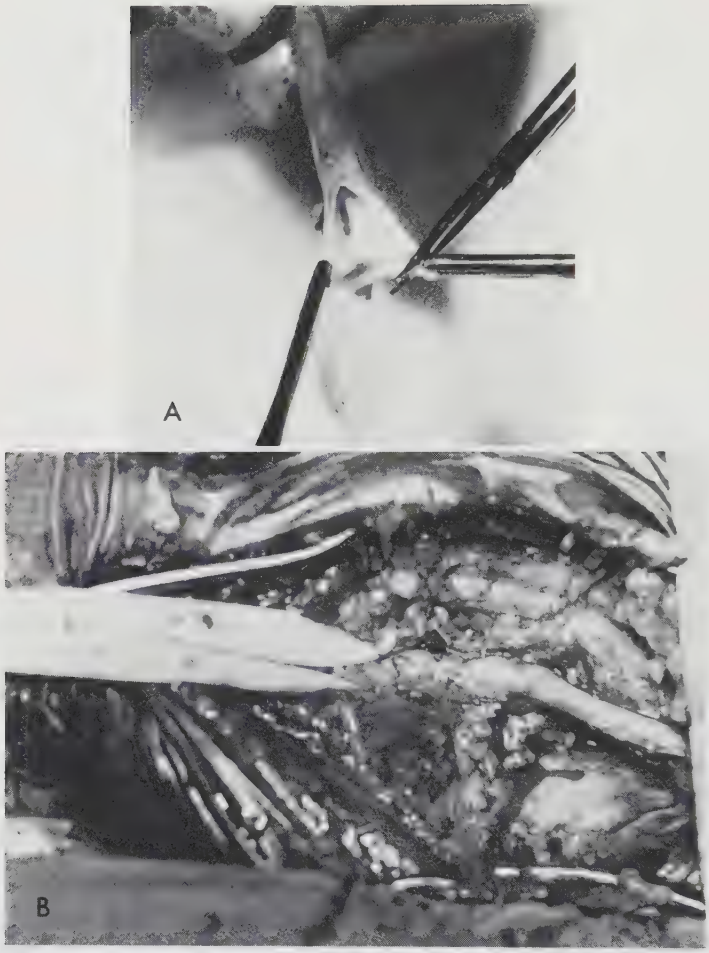


Figure 4. *A*, The vein graft has been split longitudinally and is being trimmed for anastomosis with the common femoral artery. *B*, A properly performed anastomosis to the common femoral artery results in no constriction of inflow into the vein graft.

relying upon profundaplasty as the sole operative procedure which has sometimes produced disappointing results in our experience (Fig. 5).

After completing the proximal anastomosis and release of clamps an on-the-table arteriogram is performed by injecting undiluted contrast medium into the common femoral artery above the proximal anastomosis of the graft. This permits the best assessment of the technical acceptability of the reconstruction that the surgeon can at present obtain. Significant abnormalities in the operative arteriogram which require surgical correction are seen in about 3 per cent of cases (Fig. 6).

Prophylactic antibiotics have not been routinely used for patients undergoing autogenous saphenous vein grafts, because infection rarely occurs when autogenous veins are used. Patients receiving plastic prostheses have received preoperative and intraoperative antibiotics, usually a cephalosporin, as prophylaxis against hospital strains of Staphylococcus aureus which have characteristically been the most common organisms involved in infections. Ordinarily, the heparin is not reversed after completion of the operative procedure. The wounds are closed without drains.

Femoro-Tibial Reconstruction

For femoro-tibial vein grafting the medial approach is used for the posterior tibial artery and the proximal peroneal artery. For the lower half of the peroneal artery it is technically much easier to resect a portion of the fibula and expose the artery immediately beneath the interosseous membrane. For the anterior tibial vein, the lateral approach

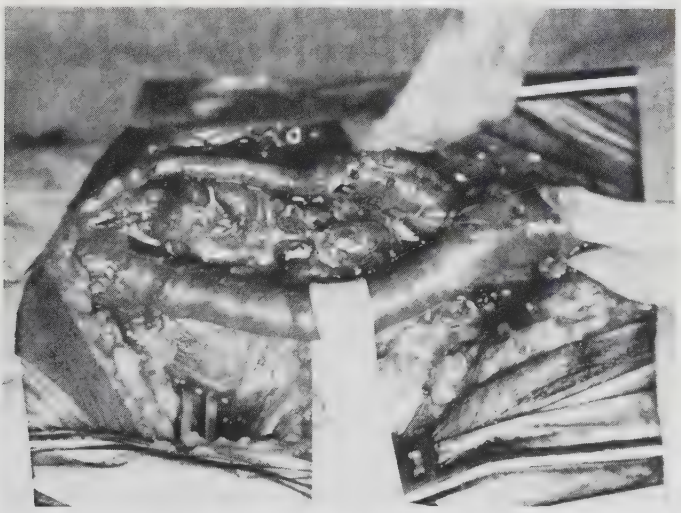


Figure 5. The origion of the vein graft is used as a profundaplasty in a patient with combined occlusion of the superficial femoral artery and stenosis of the origin of the profunda femoris artery. The first portion of the vein graft has been utilized as a patch graft over the origin of the profunda femoris artery, which is somewhat distorted because of traction on the occluded superficial femoral artery to expose the graft. The vein graft then takes off from the end of the profundaplasty.

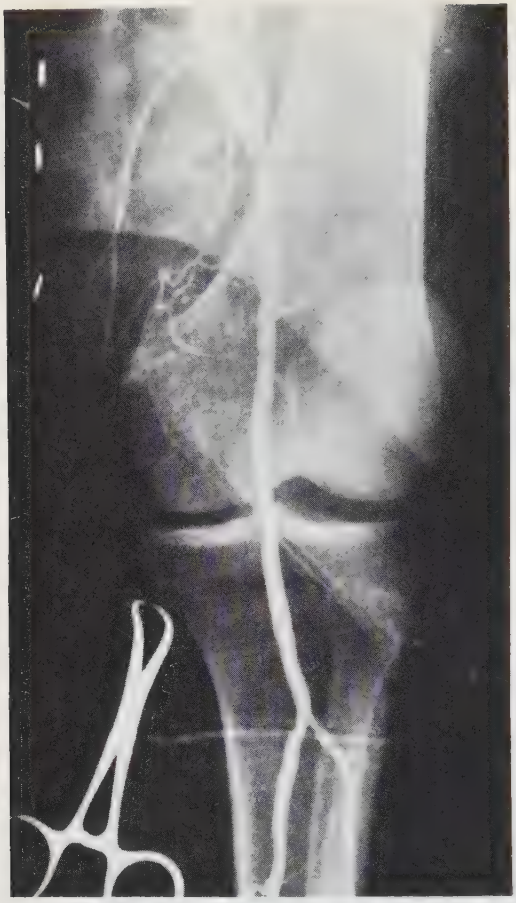


Figure 6. Irregularity at the distal end of a femoral popliteal graft is shown on an arteriogram taken intraoperatively. This lesion requires operative correction.

is necessary with retraction of the muscles in the anterior compartment, exposing the artery lying just anterior to the interosseous membrane.

Vein grafts are used exclusively for tibial artery reconstructions and we have, as in the case of femoro-popliteal reconstructions, preferred to tunnel the grafts in roughly anatomical position (Fig. 7). In some grafts to the distal posterior tibial artery a subcutaneous position of the graft may be preferable because the graft appears to have a somewhat straighter course. In tunneling the vein graft into the anterior compartment it is wise to make a large hole in the interosseous membrane so that the edge of the membrane cannot constrict the graft.

For performing tibial vessel anastomoses ocular loops are helpful. Clamps are never applied to the tibial vessels; forward and backbleeding are controlled with doubly looped strands of silastic. The suture material is 6-0 polypropylene and considerable care is taken to place all the sutures in the distal end of the anastomosis before pulling the suture tight. Again the outflow of the graft is calibrated with coronary artery dilators prior to completion of the anastomosis, and on-the-table

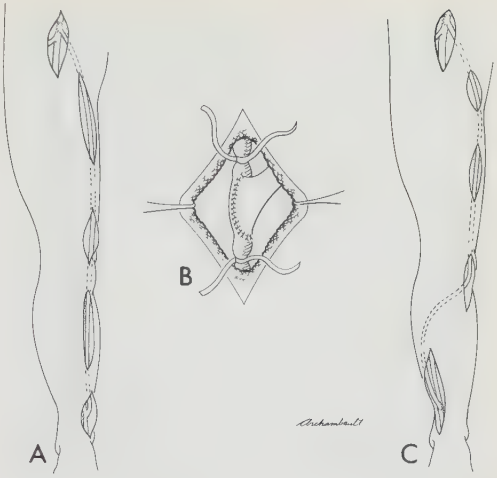


Figure 7. The technique for grafting of the femoral-tibial vein is shown. *A*, A common femoral to distal posterior tibial graft is illustrated. *B*, The proper technique of vessel control and anastomosis is outlined (see text). *C*, In a femoral-to-anterior tibial vein graft, the graft passes through the interrosseous membrane to reach the anterior tibial artery.

angiography is essential to ensure the technical adequacy of these small caliber anastomoses (Fig. 8). It is also essential for the success of femoro-tibial reconstruction for the tibial vessel into which the graft is placed to be of normal or nearly normal consistency and connected directly to the vascular arch of the foot. If this cannot be determined by preoperative angiography, a direct on-the-table angiogram may be necessary before electing to perform a tibial vessel graft.

Some patients with distal popliteal artery occlusion have, on angiography, an isolated segment of popliteal artery with superficial femoral occlusion above and a distal popliteal artery occlusion below with collateral vessels leading in and out of the isolated artery segment as the only inflow and outflow. The surgeon may be faced with the choice of placing the distal anastomosis of a vein graft in the isolated popliteal artery segment or in one of the tibial vessels in the calf or both. Use of the isolated popliteal artery segment rather than a tibial vessel for the distal anastomosis is preferred because such procedures have a higher success rate, and are technically easier to perform (Fig. 9).[8] However, in patients with marked ischemic tissue loss in the foot, particularly in those with diabetes, it is perhaps better to follow the procedure recently reported by Edwards which consists of bypasses both to the isolated popliteal segment and to a tibial vessel with a sequential vein graft.[11]

If sufficient length of good quality vein is not available for a bypass to reach from the appropriate site of distal anastomosis up to the common femoral artery, compromise may be necessary with respect to the site of the proximal anastomosis. If the superficial femoral artery is patent in the upper portion of the thigh it is an appropriate place for the proximal anastomosis. In some instances, an unsuitable portion of the vein may be used as a patch graft for an endarterectomy of the superficial femoral artery, using the remaining more normal portion of the vein for a conventional vein bypass beginning at the distal end of the patch graft (Fig. 10).

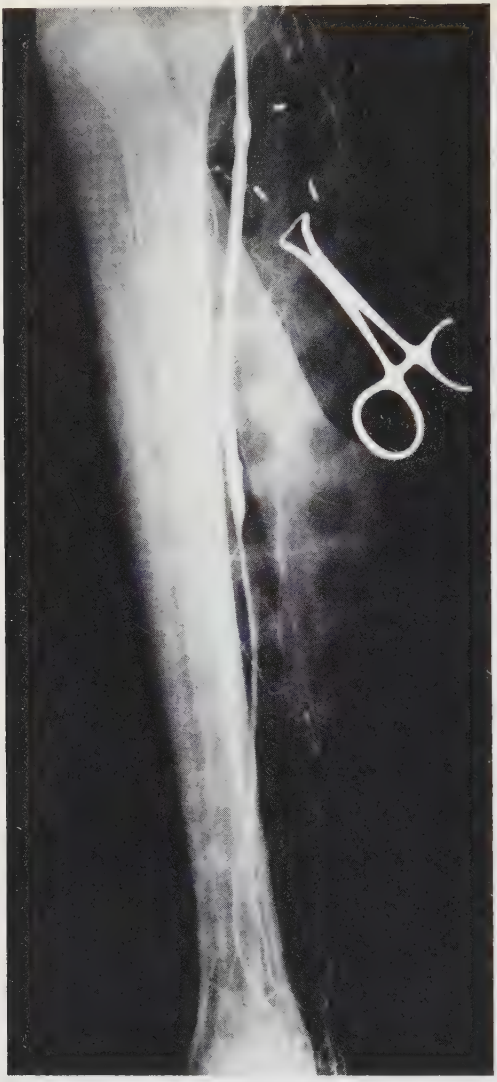


Figure 8. An on-the-table arteriogram illustrates a technically adequate vein graft to the midperoneal artery. The medial approach was used.

We have also not hesitated to use the profunda femoris as a site for proximal anastomosis of a vein graft when vein length is a problem.[4, 34] Another possibility is a composite autogenous vein graft with the two veins sutured end-to-end. While such a composite graft is theoretically attractive, these grafts have not functioned as well as single vein grafts in our experience. We have had poor results with composite Dacron and vein and PTFE and vein grafts and have abandoned this technique. We have, however, had success in a few instances with a technique described by DeLaurentis and Friedmann

Figure 9. An arteriogram of a vein graft from the left common femoral to an isolated popliteal artery segment taken three years after insertion of the graft shows the graft continuing to function well despite the relatively small size of the isolated popliteal artery segment.

which consists of a double bypass utilizing a plastic graft to an endarterectomized segment of popliteal artery above the knee, and a second bypass of vein from this same segment to a distal site.[9] As noted above, when no suitable vein is available we at present prefer a PTFE graft.

Figure 10. The upper end of a vein graft is used as a patch graft over the first portion of the superficial femoral artery in order to allow a lower take off of the vein graft and ensure a graft of adequate caliber.

POSTOPERATIVE MANAGEMENT

We believe that healing of wounds takes place more promptly if the patients are permitted to remain in bed for three to four days before beginning ambulation. In order to prevent phlebitis during this period, 5,000 units of heparin are administered subcutaneously every 12 hours until ambulation is begun. In these patients edema almost invariably develops upon placing the leg in a position of dependency. Therefore, a light-weight elastic stocking is routinely prescribed, to be worn whenever the patient is out of bed for the first six to eight weeks after surgery.

RESULTS

We have recently reported a personal experience with 111 femoropopliteal bypass vein grafts performed as the initial operation for limb salvage in 101 patients.[23] None of these patients had concomitant or subsequent aorto-iliac reconstructions. The five year cumulative graft patency by the life table method in these patients was 78 per cent and the five year cumulative limb salvage rate was 89 per cent. Over the same period of time 44 femoro-tibial vein grafts were performed in 41 patients with a five year cumulative graft patency of 55 per cent and a limb salvage rate of 73 per cent. During the same period 49 femoropopliteal grafts were performed in 41 patients for the primary indication of intermittent claudication. These included Dacron grafts, PTFE grafts, and vein grafts. The five-year cumulative patency rate of the grafts performed for claudication was 88 per cent and no patient in this group lost a limb within five years of arterial surgery. The operative mortality rate was slightly more than 2 per cent for femoropopliteal and femoro-tibial vein grafts performed for limb salvage and was zero for patients operated upon for claudication. The five-year survival for the entire patient group operated upon for limb salvage because of advanced femoro-popliteal-tibial occlusive disease is, however, disappointingly low.[23] Only about 50 per cent of these individuals survive for five years, the majority dying of complications of arteriosclerosis, usually coronary artery disease.

Although it is clear from the figures quoted above that when femoro-popliteal or femoro-tibial grafts are performed for limb salvage the long-term salvage exceeds graft patency by about 10 per cent, the majority of patients in this group whose grafts fail are again in jeopardy of major amputation. Our experience with these individuals suggests that a further attempt at arterial reconstruction is worthwhile. Over the past decade we have performed 45 reoperative procedures for arterial reconstruction in 28 of these patients with a four-year graft and limb salvage rate of 56 per cent. The operative mortality rate again was approximately 2 per cent.

The causes of immediate femoro-popliteal vein graft failure appear to be almost invariably technical including the attempted use of a prosthetic graft in a patient whose outflow is poor. Poor outflow appears rarely to be the cause of early vein graft failure, however,[24, 28] and with the use of on-the-table angiography and correction of techni-

cal deficiencies at the time of the initial operation, we have reduced our early vein graft failure rate to less than 5 per cent over the past ten years.[23]

Most so-called "late" vein graft failures occur within the first six months after implantation. The cause of graft failure in this period commonly appears to be intimal hyperplasia of the graft itself which in turn may be related to intimal injury at the time of harvesting of the vein graft,[13, 19, 32] and if experimental evidence is to be believed,[1, 19, 32] failure might be reduced if chilled autogenous blood were used to prepare and dilate the graft. However, we have been loath to adopt this technique for fear of leaving fibrin deposited on the venous valves during the course of vein graft preparation and thus possibly inducing early graft thrombosis. We have compromised by utilizing a chilled isotonic electrolyte solution at a physiologic pH containing heparin. Whether or not the use of antiplatelet agents in the perioperative period would further reduce the incidence of intimal hypertrophy after vein grafting is not clear at this time. The common cause of truly late failure (beyond two years) of fabric and vein grafts appears to be advancing arteriosclerotic disease in the host vessels. A characteristic lesion following implantation of a fabric graft appears to be a hyperplastic intimal lesion in the host artery, just distal to the distal anastomosis of the graft, which ordinarily advances slowly over several years.

The results reported above are not as discouraging as some reported in the recent surgical literature which have led a number of experienced surgeons to adopt a skeptical attitude toward the usefulness of femoro-popliteal reconstruction for either claudication or limb salvage.[25, 31, 36, 39] However, with careful attention to technical detail and the liberal use of on-the-table angiography, we are convinced that quite acceptable early and long-term results are possible with femoropopliteal and femoro-tibial reconstruction.[6, 16, 22, 23, 29, 30, 33] It seems likely that the patient who is in danger of major amputation or who has developed disabling claudication on the basis of femoro-popliteal-tibial occlusive disease will find that long-term relief of disability with minimal operative risk is preferable to continued ischemic symptoms and/or major amputation.

REFERENCES

1. Abbott, W. M., Wieland, S., and Austen, W. G.: Structural changes during preparation of autogenous venous grafts. Surgery, *76*:1031–1060, 1974.
2. Bernhard, V. M., Militello, J. M., and Geringer, A. M.: Repair of the profunda femoris artery. Am. J. Surg., *127*:676–679, 1974.
3. Burnham, S. J., Flanigan, D. P., Goodreau, J. J., et al.: Nonvein bypass in below-knee reoperation for lower limb ischemia. Surgery, *84*:417–424, 1978.
4. Buxton, B., Reeves, L., and Roberts, A. K.: Distal profunda femoris to popliteal artery bypass for patients with a short length of long saphenous vein. Surgery, *83*:245–247, 1978.
5. Clayson, K., Edwards, S., Terry, A., et al.: Arm veins for peripheral arterial reconstruction. Arch. Surg., *111*:1276–1280, 1976.
6. Cutler, B. S., Thompson, J. E., Kleinsasser, L. J., et al.: Autologous saphenous vein femoropopliteal bypass: Analysis of 298 cases. Surgery, *97*:325–331, 1976.
7. David, T. E., and Drezner, A. D.: Extended profundoplasty for limb salvage. Surgery, *84*:758–762, 1978.
8. Davis, R. C., Davies, W. T., and Mannick, J. A.: Bypass vein grafts in patients with distal popliteal artery occlusion. Am. J. Surg., *29*:421–425, 1975.

9. DeLaurentis, D. A., and Friedmann, P.: Arterial reconstruction about and below the knee, Am. J. Surg., *21*:121–392, 1971.
10. DeWeese, J. A., and Rob, C. G.: Autogenous venous grafts ten years later. Surgery, *82*:775–784, 1977.
11. Edwards, W. S., Gerety, E., Larkin, J., et al.: Multiple sequential femoral tibial grafting for severe ischemia. Surgery, *80*:722–728, 1976.
12. Fry, P. D., and Robertson, M. E.: Initial experience with the polytetrafluoroethylene graft for limb salvage. Am. J. Surg., *136*:193–197, 1978.
13. Fuchs, J. C. A., Mitchener, J. S., and Hagen, P.: Postoperative changes in autologous vein grafts. Ann. Surg., *188*:43–48, 1978.
14. Hershey, F. B., and Auer, A. I.: Extended surgical approach to the profunda femoris artery. Surg. Gynecol. Obstet., *138*:88–90, 1974.
15. Imparato, A. M., Kim, G. E., Davidson, T., et al.: Intermittent claudication: Its natural course. Surgery, *78*:795–799, 1975.
16. Imparato, A. M., Kim, G. E., Madayag, M., et al.: The results of tibial artery reconstruction procedures. Surg. Gynecol. Obstet., *138*:33–38, 1974.
17. Inahara, T., and Toledo, A. C.: Endarterectomy of the popliteal artery for segmental occlusive disease. Ann. Surg., *188*:43–48, 1978.
18. Kakkar, V. V.: The cephalic vein as a peripheral vascular graft. Surg Gynecol Obstet., *128*:551–556, 1968.
19. Karayannacos, P. E., Hostetler, J. R., Bond, M. G., et al.: Late failure in vein grafts: Mediating factors in subendothelial fibromuscular hyperplasia. Ann. Surg. *187*:183–188, 1978.
20. Kunlin, J.: Le traitment de l'ischemia arteritique par la greffe veinuse longue. Rev. Chir. Orthop., *70*:206, 1951.
21. Leather, R. P., Shah, D. M., and Karmody, A. M.: The use of extended profundaplasty in limb salvage. Am. J. Surg., *136*:359–362, 1978.
22. LoGerfo, F. W., Corson, J. D., and Mannick, J. A.: Improved results with femoropopliteal vein grafts for limb salvage. Arch. Surg., *112*:567–570, 1977.
23. Maini, B. S., and Mannick, J. A.: Effect of arterial reconstruction on limb salvage. Arch. Surg., *113*:1297–1304, 1978.
24. Mannick, J. A., and Jackson, B. T.: Hemodynamics of arterial surgery in atherosclerotic limbs. I. Direct measurement of blood flow before and after vein grafts. Surgery, *59*:712–720, 1966.
25. Martin, C., and Foster, J. H.: Factual palliation with femoropopliteal bypass for salvage. J. Surg. Res., *18*:215–220, 1975.
26. Martin, P., and Jamieson, C.: The rationale for and measurement after profundaplasty. SURG. CLIN. N. AM., *54*:95–109, 1974.
27. McAllister, F. F.: The fate of patients with intermittent claudication managed nonoperatively. Am. J. Surg., *132*:593–595, 1976.
28. Mundth, E. D., Darling, R. C., Moran, J. M., et al.: Quantitative correlation of distal arterial outflow and patency of femoropopliteal reversed saphenous vein grafts with intraoperative flow and pressure measurements. Surgery, *65*:197–206, 1969.
29. Naji, A., Barker, C. F., Berkowitz, H. D., et al.: Femoro-popliteal vein grafts for claudication: Analysis of 100 cases. Ann. Surg. *188*:79–82, 1978.
30. Naji, A., Chu, J., McCombs, P. R., et al.: Results of 100 consecutive femoropopliteal vein grafts for limb salvage. Ann. Surg., *188*:1–15, 1978.
31. O'Donnell J. A., Brener, B. J., Brief, D. K., et al.: Realistic expectation for patients having lower extremity bypass surgery for limb salvage. Arch. Surg., *112*:1356–1363, 1977.
32. Ramos, J. R., Berger, K., Mansfield, P. B., et al.: Histologic fate and endothelial changes of distended and nondistended vein grafts. Ann. Surgery, *183*:205–228, 1976.
33. Reichle, F. A., and Tyson, R. R.: Comparison of long-term results of 364 femoropopliteal or femorotibial bypasses for revascularization of severely ischemic lower extremities. Ann. Surg., *182*:449–454, 1975.
34. Stabile, B. E., and Wilson, S. E.: The profunda femoris-popliteal artery bypass. Arch. Surg., *113*:1297–1304, 1978.
35. Stoney, R. J.: Ultimate salvage for the patient with limb-threatening ischemia. Am. J. Surg., *136*:228–232, 1978.
36. Stoney, R. J., James D. R., and Wylie, E. J.: Surgery for femoropopliteal atherosclerosis: A reappraisal. Arch Surg., *103*:548–553, 1971.
37. Veith, F. J., Moss, C. M., Fell, S. C., et al.: Comparison of expanded polytetrafluoroethylene and autologous saphenous vein grafts in high risk arterial reconstructions for limb salvage. Surg. Gynecol. Obstet., *147*:749–752, 1978.
38. Veith, F. J., Moss, C. M., Fell, S. C., et al.: Expanded polytetrafluoroethylene grafts in reconstructive arterial surgery. J.A.M.A., *240*:1867–1869, 1978.

Peter Bent Brigham Hospital
721 Huntington Ave
Boston, Massachusetts 02115

Aortic Aneurysm: Current Status of Surgical Treatment

E. Stanley Crawford, M.D., Arthur E. Palamara, M.D.,† Salwa A. Saleh, M.D.,‡ and John O. F. Roehm, Jr., M.D.§*

Significant progress has been made during recent years in the surgical treatment of aortic aneurysm. Surgical techniques have been developed and improved for successful treatment regardless of the type, location, and extent of the disease, and operative mortality has steadily decreased and long-term survival has been favorably influenced.[13, 16, 30, 33, 39] Contributing to this progress have been advances in concepts of the disease and numerous measures which have been introduced in support of operation. All aneurysms of the aorta are now susceptible to and successfully treated by operation; however, the indications, objectives, and results of therapy vary with the type and location of the aneurysm. This report emphasizes current concepts and principles.

SELECTION OF PATIENTS FOR OPERATION

Acute Dissecting Aortic Aneurysms

There is now a consensus among surgeons that all acute dissections involving the ascending aorta are best treated by operation to relieve or prevent aortic insufficiency and rupture into the heart or pericardium.[1, 2, 20, 23, 27]

It is also agreed that acute dissections arising in and involving the descending thoracic aorta and associated with continued dissection, occlusion of the distal vessels, and aortic dilatation and rupture are preferably treated by operation. Earlier in the experience in the treatment of aneurysms at this level, medical treatment (hypotensive medical management) was considered to be preferable by some in patients without complications because there was a higher earlier survival rate with

From Baylor College of Medicine and The Methodist Hospital, Houston, Texas

*Professor of Surgery
†Cardiovascular Surgical Fellow
‡Clinical Instructor in Anesthesiology
§Clinical Associate Professor of Radiology

this form of therapy.[35, 36] More recently, a combination of medical and surgical treatment has become popular for these patients.[1, 2, 30, 39] Hypertension and the dissecting process are first stabilized by medical treatment except in patients with rupture, who are immediately submitted to operation. Medical treatment is continued after operation as it would have been had medical treatment alone been chosen. This approach has been justified on the basis of a steady decline in operative mortality and a 60 percent three year cumulative survival rate in contrast to a rate of 30 percent in medically treated patients.[30, 39]

Chronic Dissecting, Chronic Traumatic, and Degenerative Aneurysms of the Thoracic Aorta

Operation is recommended in the treatment of these lesions to control pain, rupture, and other complications regardless of their location and extent. The same principles apply to those lesions that involve both the thoracic and the abdominal aorta including the segment from which the visceral vessels arise.

Abdominal Aortic Aneurysms

It is agreed that abdominal aortic aneurysms regardless of their extent and size are preferably treated by operation except in patients with small aneurysms, less than 5 cm in diameter. There is increasing evidence, however, that such small aneurysms are not benign. Experienced surgeons see rupture in small aneurysms and a recent study of autopsies in 473 patients who died with untreated abdominal aortic aneurysms showed death was caused by rupture of the aneurysm in 118 (25 per cent) patients.[14] Moreover, the relationship of rupture and death to the diameter of the aneurysm was 9.5 per cent in those with a diameter of 4 cm or less, 23 per cent in those with a diameter of 4 to 5 cm, 46 per cent in those with a diameter of 7 to 10 cm, and 61 per cent in those with diameters greater than 10 cm. Consequent to these experiences, operation is being advised for all aneurysms including small ones in patients who would tolerate operation well and for enlarging, large, symptomatic, and ruptured aneurysms in the poorer risk patient.

CURRENT METHODS OF STUDY

Thoracic aortic aneurysms may be suspected from widening of the mediastinum or mediastinal masses in plain roentgenograms; however, precise diagnosis of the disease, its nature, location, and extent can be determined only by aortography, preferably performed by power injection of concentrated media through an intra-aortic catheter and multiple exposures in the anteroposterior, oblique, and lateral positions. Computed axial tomography and ultrasonic techniques are not sufficiently developed to be of practical clinical value at this time in the study and treatment of thoracic aortic aneurysms.[32]

Diagnosis of abdominal aortic aneurysm may be made by palpation, by plain roentgenograms in the anteroposterior and lateral positions, by aortography, and by ultrasonographic techniques by experienced technicians with proper equipment.[4, 7, 31] The accuracy of diagnosis varies with each method (90 per cent by aortography, and virtually 100 per cent by ultrasonography). The size of an aneurysm is best estimated by plain roentgenograms when visualized and by ultrasonography.[7] Aortography by the previously mentioned technique is best suited for determining the extent of disease, the involvement of visceral vessels, and the presence of associated disease of visceral, iliac, and more distal arteries, for aid in the design of the operation.[8]

REDUCTION OF RISK FROM ASSOCIATED DISEASE

A major factor in the improvement in survival rates following operation has been identification and appropriate treatment of diseases that pose special risks, including coronary artery and carotid artery disease. The most common disabling and lethal complications in patients with unstable coronary artery disease and active cerebrovascular disease are myocardial infarction in the former and stroke in the latter. We have performed coronary artery bypass grafts first and then aortic operation at a later date in over 100 patients with no deaths occurring after aortic operation.[11] Similarly, carotid endarterectomy has been performed before or at the time of other operation in 128 patients with survival in most. Of special significance is the fact that permanent stroke did not occur in any of these patients. Other simultaneously performed secondary operations that have reduced risks in the treatment of abdominal aortic aneurysm are cholecystectomy, duodenal and gastric ulcer operations, and repair of hiatus hernia.

PROPHYLACTIC ANTIBIOTICS

Prophylactic antibiotics have been given according to various protocols by most surgeons for years in patients submitted to cardiac and vascular operations. There was general clinical agreement that such treatment reduced the incidence of infection complicating operation. A recently published well-controlled prospective double blind study firmly establishes this clinical impression.[22] Cefazolin given prophylactically, "on call," during long operations, and four times at six-hour intervals after operation was associated with an incidence of infection in the wound, about the graft, or in the blood stream in 0.9 per cent of patients. Sham injections of saline were associated with infection in 6.8 per cent. We give Staphcillin or Keflin prophylactically 12 hours and two hours before operation, during operation, and every six hours for three days or until all indwelling tubes are removed.

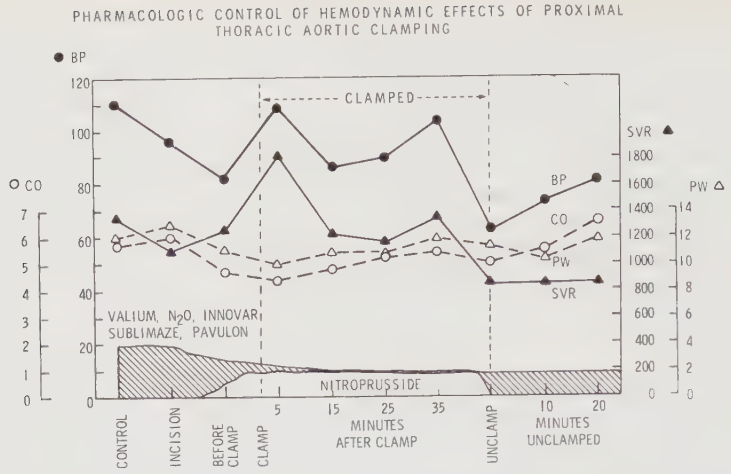


Figure 1. Pharmacologic control is shown in blood pressure (BP), cardiac output (CO), pulmonary wedge pressure (PW), and systemic vascular resistance (SVR) readings obtained in 12 patients (average values) intermittently before, during, and after clamping of the proximal descending thoracic aorta without shunt.

GENERAL SUPPORTIVE MEASURES

Anesthesia is obtained by drugs that do not depress the myocardium, and relaxation is encouraged with muscle relaxants. Intra-arterial blood pressure, electrocardiography, and central venous pressure are monitored continuously in all patients undergoing aortic operation. Blood gases, serum electrolytes, and plasma colloidal osmotic pressures are measured every 30 minutes during operation. Urine output is measured continuously by indwelling catheter. Appropriate treatment is given for the abnormalities detected. Cardiac output, pulmonary wedge pressure, and systemic vascular resistance are measured intermittently by Swan-Ganz catheter in patients before, during, and after clamping of the descending thoracic aorta. These measurements and arterial blood pressure are maintained at desirable levels during the period of aortic clamping in order to avoid cardiac strain, and to provide maximum collateral circulation during operation for aneurysm involving the descending thoracic aorta, since we do not use bypass shunts (Fig. 1).

Electrolyte solutions begun before operation are maintained during the procedure maintaining the central venous pressure between 7 and 10 mm of water. The first liter is given as 5 per cent dextrose and Ringer's lactate solution and the remainder as Ringer's lactate solution. Mannitol solution, 25 gm, is given after induction of anesthesia. Blood replacement by the component technique using packed cells, fresh frozen plasma, and platelets provides better utilization of available blood and significantly reduces the incidence of intraoperative coagulopathy. As blood replacement becomes necessary, 250 cc of packed red blood cells to which 100 cc of sodium chloride, U.S.P., have been added are given and continued as necessary. A unit of fresh frozen plasma is given

after the third transfusion and is then alternated with two units of red cells. The routine is altered when greater blood loss is expected, that is, 8 to 15 units are given during operation for ruptured aneurysm and during replacement of thoracic and thoraco-abdominal aortic aneurysms. A unit of albumin and two units of fresh frozen plasma may be given before or at the start of operation. Transfusion is continued as above and 10 to 16 units of fresh platelets are given when flow is restored through the graft.

Operation should be delayed 2 to 4 days in patients taking Coumadin until prothrombin time is normal. If emergency operation is necessary, Aquamephyton and 2 to 4 units of fresh frozen plasma should be given beforehand, and administration of plasma should be continued until prothrombin times are within normal range. Amicar (5 gm per 20 cc in 250 cc of saline over a one-hour period) should be given to correct disturbances of fibrinolysis, and fibrinogen should be corrected by giving cryoprecipitate. In our experience, four to six units have been adequate.

ANEURYSMS OF THE ASCENDING AORTA

Current methods of treating dissecting aortic aneurysms involving the ascending aorta are diagrammed in Figures 2 to 4. Choice of method depends upon the origin of the dissection, the presence or absence of

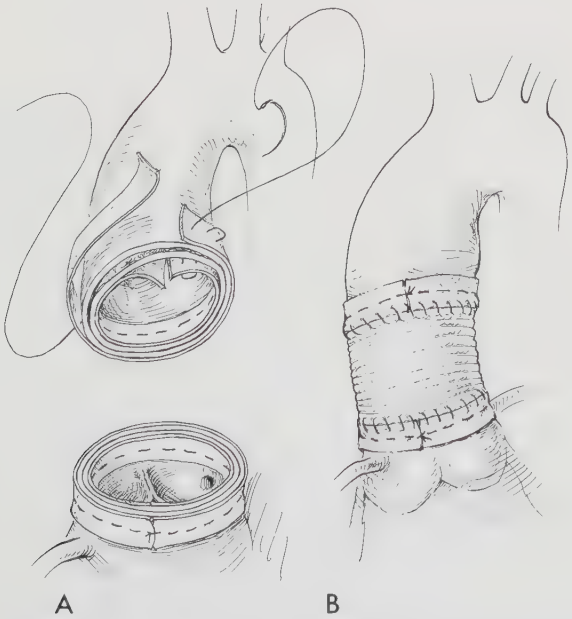


Figure 2. A method of repair of dissecting aneurysms in the ascending aorta does not involve replacement of valves. *A*, A segment of the ascending aorta is removed and this aortic wall is reconstructed using Dacron felt. *B*, The method of insertion of the woven Dacron graft is illustrated.

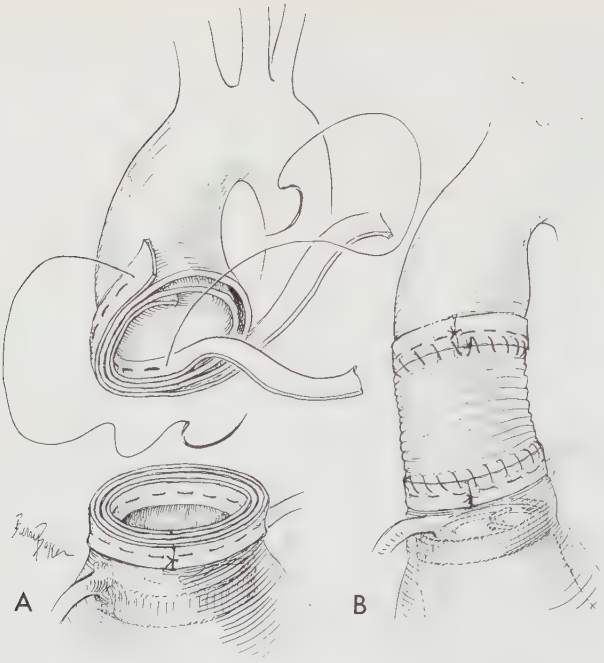


Figure 3. A second method of repair of dissecting aneurysm in the ascending aorta employs valve replacement. *A*, A segment of the ascending aorta is removed and the remaining aorta is repaired as shown in Figure 1. *B*, The aortic valve is then replaced and the graft is inserted.

aortic insufficiency, and disruption of the aorta near the annulus as indicated by the presence of hematoma, bleeding at the site, blood in the pericardium, or thinning of the aorta. A graft of woven Dacron is used in all cases to interrupt the dissection, to replace the origin of dissection, to replace the ruptured section of aorta, and to take tension off the suture lines of the repair.

In uncomplicated cases without aortic insufficiency, and in those patients with aortic insufficiency but in whom the tough inner layer is intact, the graft is inserted after the inner and outer lumens are reapposed both proximally and distally using Dacron felt (see Fig. 2). Blood flow is thus directed distally into the normal lumen, reducing the pressure in the distal false lumen around the origins of the branches, permitting return of flow into these branches and arresting the dissecting process. Proximally, dissection is interrupted and aortic insufficiency is relieved in most cases by resuspension of the valvular apparatus. In the presence of aortic insufficiency and severe laceration or destruction of the inner layer, both the valvular apparatus and the aortic valve are excised and a mechanical valve is inserted (Figs. 3 and 5).

In patients with advanced aortic root injury with leakage, disruption, or excessive weakening of the aortic wall, the entire aortic root is replaced with a composite graft-valve prosthesis which is sutured prox-

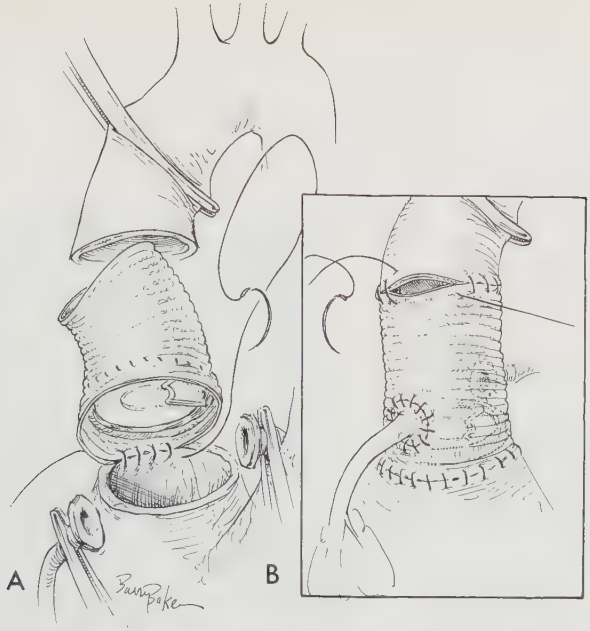


Figure 4. A third method involves complete replacement of the aortic root to treat dissecting aortic aneurysm. *A*, The aortic valve and proximal aorta are removed and a composite valve graft is sutured to the annulus. *B*, The coronary origins are reattached to the graft by suturing the button of the aorta from which the arteries arose to openings made in the graft.

imally directly to the aortic annulus. The coronary arteries are reattached either by vein graft when dissection extends out into the coronary arteries or by direct anastomosis of coronary artery orifice by various techniques, one of which is shown in Figure 4. We have successfully employed these techniques in the treatment of 15 patients; however, one patient with aortic insufficiency treated by the method shown in Figure 2 required reoperation at two weeks to insert an aortic valve to correct recurrent aortic insufficiency. A second patient treated by this method shown in Figure 3 required reoperation at three months to correct a false aneurysm occurring at the proximal suture line.

Excisional therapy has been employed over the past 20 years in 22 patients with degenerative fusiform aneurysms of the ascending aorta. Severe chronic aortic insufficiency was present in 19, requiring aortic valve replacement. Aortic continuity was restored by graft in 14 patients and end-to-end anastomosis after excision in eight patients (Fig. 6). Death occurred in half of the former, from bleeding in two and advanced myocardial and coronary artery disease in five. All patients treated by end-to-end anastomosis survived, demonstrating the feasibility and safety of primary anastomosis.[34] Surgeons with more experience in recent years have achieved a much higher incidence of success using composite graft-valve prostheses with reattachment of the coronary arteries and sealing of the region of operation inside the reconstructed aneurysmal wall to prevent bleeding.[3, 18, 24, 27]

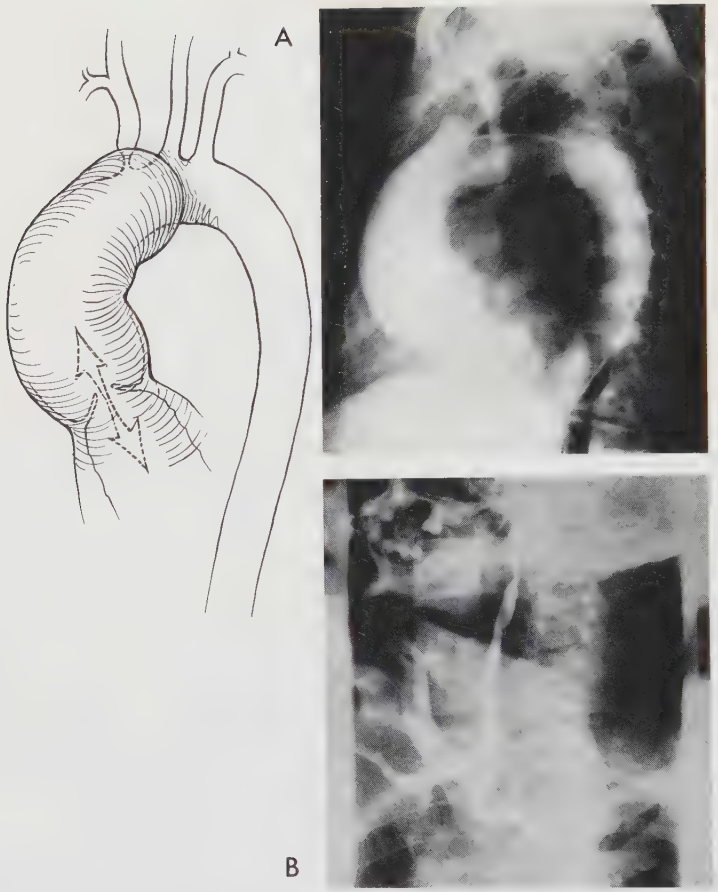


Figure 5. A patient with an acute dissecting aortic aneurysm, aortic insufficiency, and weakness in the left side of the body was treated by the insertion of a graft and replacement of the valve. *A* and *B*, The extent of disease includes compression of the innominate and right common carotid arteries.

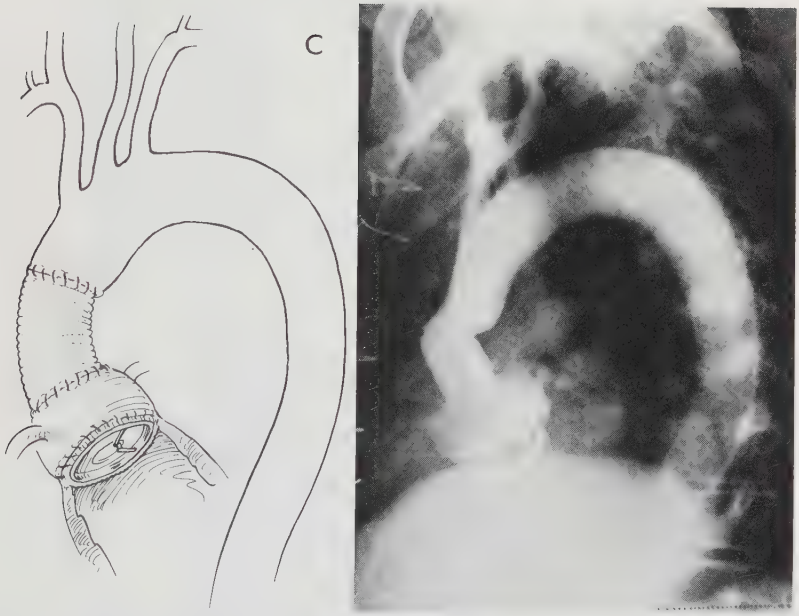


Figure 5 (*Continued*). *C*, The methods of treatment and correction of both the aortic insufficiency and the compression of the great vessels are shown.

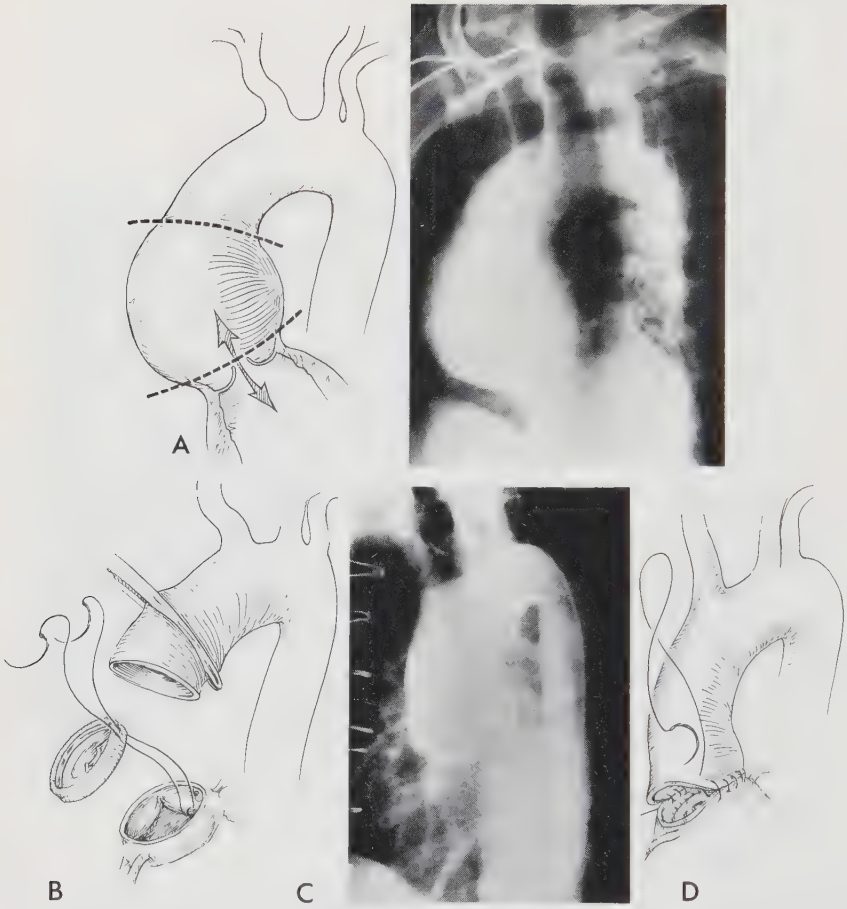


Figure 6. *A, B,* and *D,* A patient with a fusiform aneurysm (cystic medial necrosis) in the ascending aorta and aortic insufficiency was treated by removal of the aneurysm with aortic end-to-end anastomosis and replacement of the valve. *C,* An aortogram after the operation showed the reconstructed aorta and the competent aortic valve.

ANEURYSMS OF THE TRANSVERSE AORTIC ARCH

The transverse aortic arch is defined as that segment of the aorta from which the brachiocephalic vessels arise. Aneurysms occurring in this region may be fusiform. They may involve the entire segment or may be more limited in extent. In our experience, the latter are usually located in the distal arch and involve the left subclavian artery. The aneurysms are frequently sacciform, involving a part of the aortic circumference. These may be located anywhere in the arch. Operative repair requires occlusion of both the aorta and one or more brachiocephalic arteries.

To prevent cardiac strain and ischemia of distant organs, temporary bypass grafts were originally used when the proximal ascending aorta was not involved.[15] When the entire ascending aorta was involved, cardiopulmonary bypass and separate perfusion of brachiocephalic vessels were employed.[6] These methods, however, can be technically difficult and reported survival rates, although variable, were limited, causing some surgeons to feel that these lesions were not often susceptible to successful operation.

Cardiopulmonary bypass, profound hypothermia, and total circulatory arrest have recently been introduced in the treatment of these lesions.[19, 29] These techniques technically facilitate replacement but certain unsolved metabolic and hematologic problems have occurred. Preliminary reports indicate a 25 per cent mortality rate, and show that excessive bleeding frequently occurs during the warm-up period. Moreover, cerebral dysfunction continues to occur in some patients either from emboli or as a result of inadequate protection during the procedure.

The authors have treated 30 patients with lesions of the transverse aortic arch during the past 20 years with survival in 26 (87 per cent). The method of treatment varied with the type, location, and extent of the disease. Aneurysms were either sacciform or fusiform, and were located in the distal arch involving the segment of aorta from which either the subclavian or the left common carotid arteries arose in 17 patients (Fig. 7). These patients were treated without perfusion techniques. Exposure was obtained by left posterolateral thoracotomy. Circulatory arrest in the region of operation was accomplished by cross clamping the aorta proximal to the left common carotid artery and distal to the left subclavian artery. The common carotid and the left subclavian arteries were clamped distally. Hemodynamics of circulation were controlled by pharmacologic agents. The lesions were removed and replaced with patches in those with sacciform aneurysms and by tube grafts in patients with fusiform aneurysms (Fig. 7). The subclavian artery was reattached by 8 mm knitted grafts in the latter. Of these 17 patients, 16 survived without complications.

The aneurysm was located more proximally in the arch in four patients. Cardiac egress and distal circulation either to the head or to both the head and the body were maintained by permanent innominate and left common carotid artery bypass in two patients and by temporary

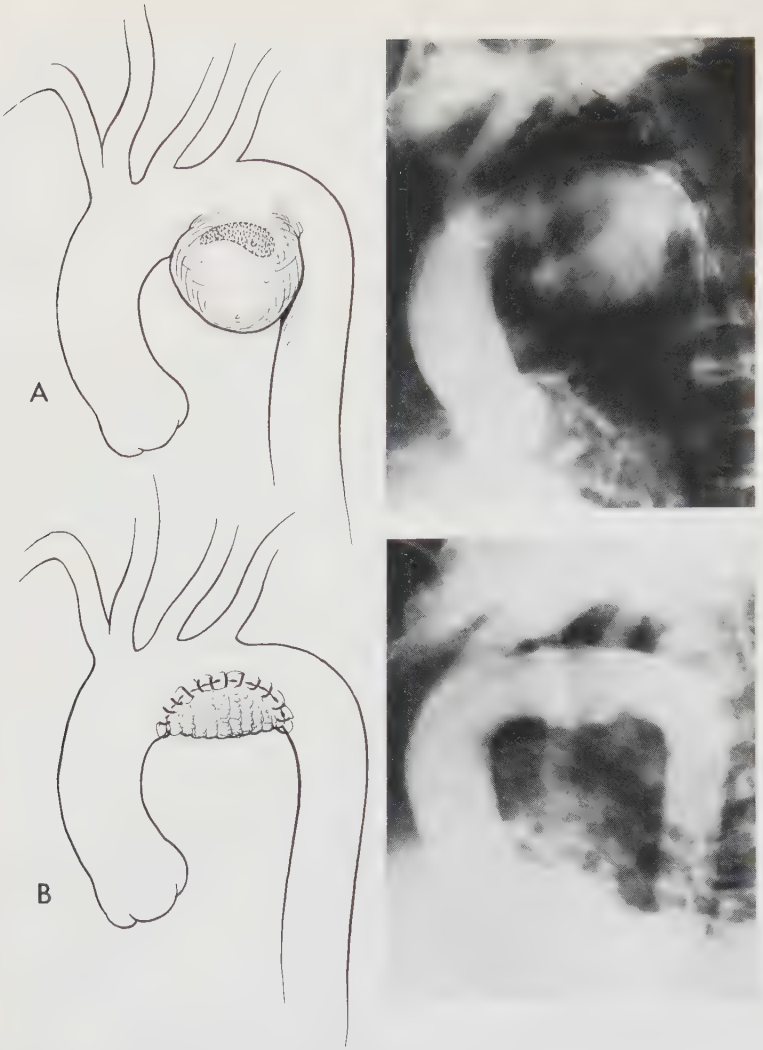


Figure 7. A sacciform aneurysm in the distal aortic arch opposite the left common carotid and subclavian arteries (*A*) was repaired with a patch graft (*B*).

bypass in three patients (Fig. 8). Of these four patients, three survived without complications.

The aneurysm involved the entire transverse arch in nine patients. In two patients, including one with total aortic involvement, the proximal ascending aorta was not involved. In the former, the arch aneurysm was successfully replaced using a temporary graft to bypass the brachiocephalic vessels and the aortic arch. The entire aorta was replaced with a graft after inserting a permanent bypass graft from the ascending aorta to the innominate, the left common carotid, and the left subclavian arteries. This patient died eight days later from pancreatitis. Cardio-

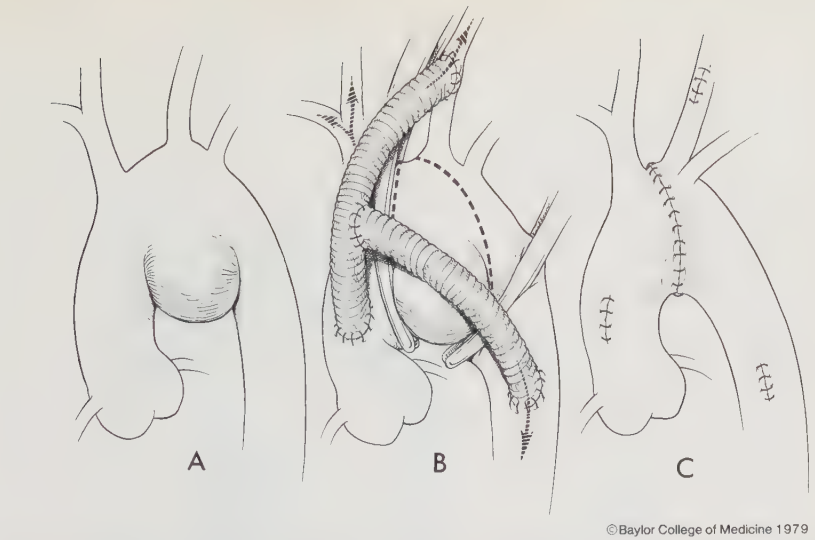


Figure 8. The method of repairing a chronic traumatic aneurysm in the middle transverse arch is shown. *A*, The location and extent of the disease are evident. *B*, Grafts and clamps are applied to control the circulation. *C*, The aorta is permanently repaired after removal of the aneurysm and the temporary Dacron grafts.

pulmonary bypass with separate brachiocephalic perfusion using various techniques was employed in the remaining patients (Figs. 9 and 10). Of these seven patients, six (85 per cent) survived operation but one had permanent neurologic sequelae. Best results were obtained with low flows (75 to 100 cc per min) and low pulse amplitude.

We feel that operation can be effectively employed as treatment for patients with aneurysms of the aortic arch, and given the magnitude of problems presented by these cases, we consider these results to be satisfactory and believe that they can be improved upon with additional experience.

ANEURYSMS OF THE DESCENDING THORACIC AORTA

Acute Dissections

The method for treating acute dissections associated with pain and occlusion of distal vessels is shown in Figure 11. Aortic resection is limited to removing the point of entry. The distal aorta is bevelled, saving the origins of all intercostal arteries, and distal false lumen closed by opposing both aortic walls between two strips of Dacron felt as a sandwich. The end of a woven tubular Dacron graft is attached proximally and then the distal end bevelled to fit the distal reconstituted aorta. The distal anastomosis is then performed, rediverting all blood flow through the normal lumen, arresting the dissection and taking pressure off origins of distal branches and permitting resumption of normal blood flow. This type of operation is very effective in restoring circulation in

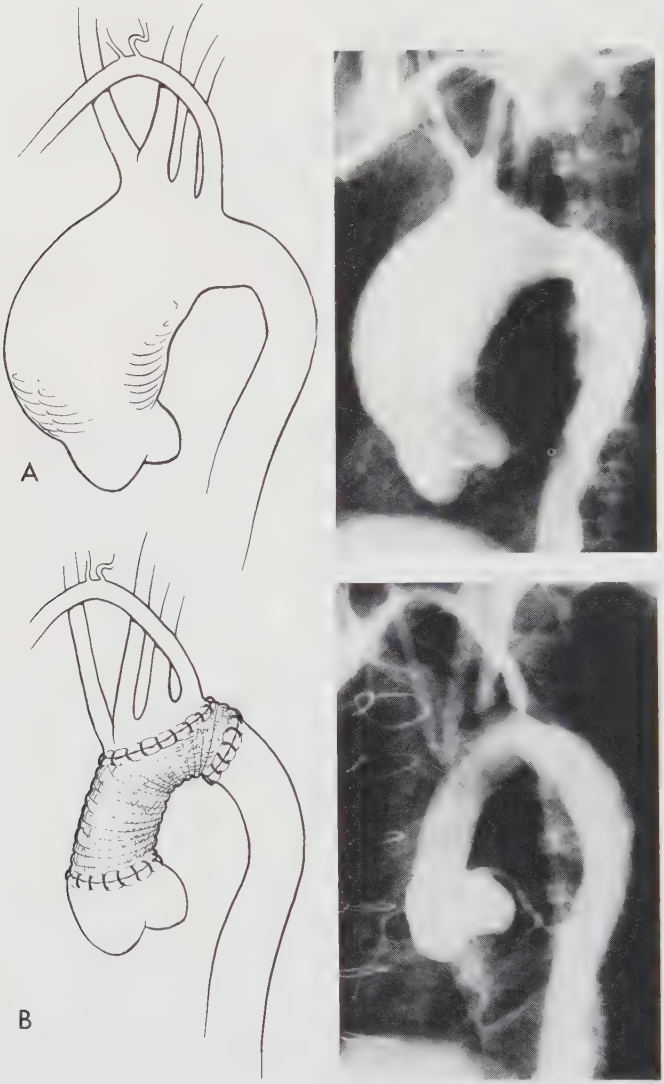


Figure 9. A patient with a fusiform aneurysm involving the entire ascending aorta and the transverse arch was treated with a replacement graft. *A*, The preoperative extent of disease is shown. *B*, After repair using cardiopulmonary bypass perfusion the aorta appeared normal.

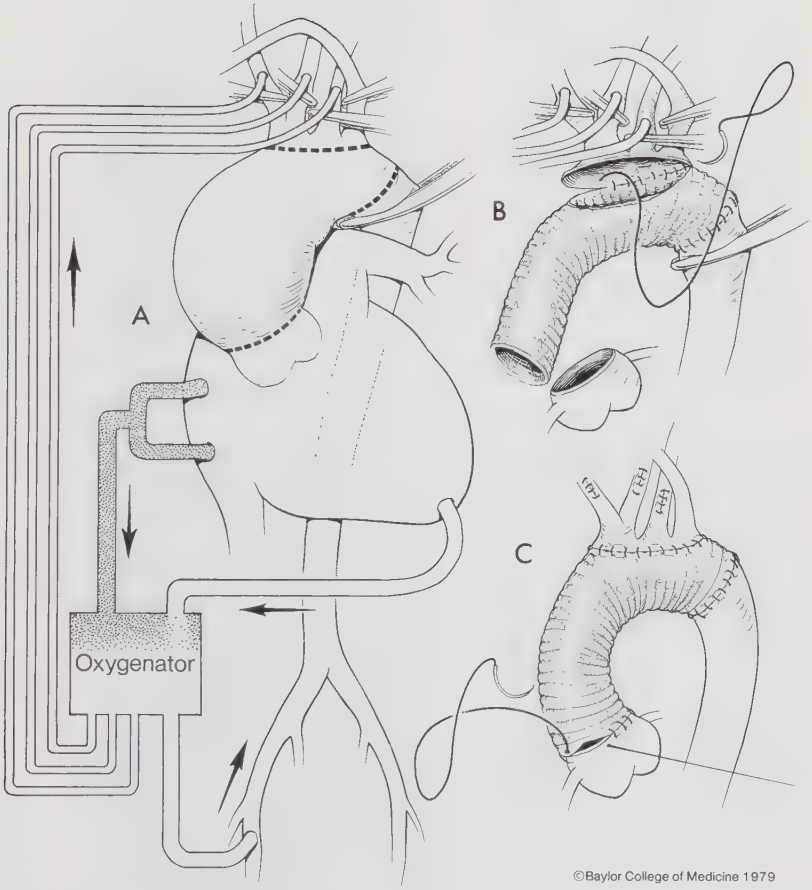


Figure 10. *A*, The patient in Figure 9 was treated with perfusion through arterial cannuli. *B*, After removal of the aneurysm the aorta was reconstructed. *C*, The graft is in place.

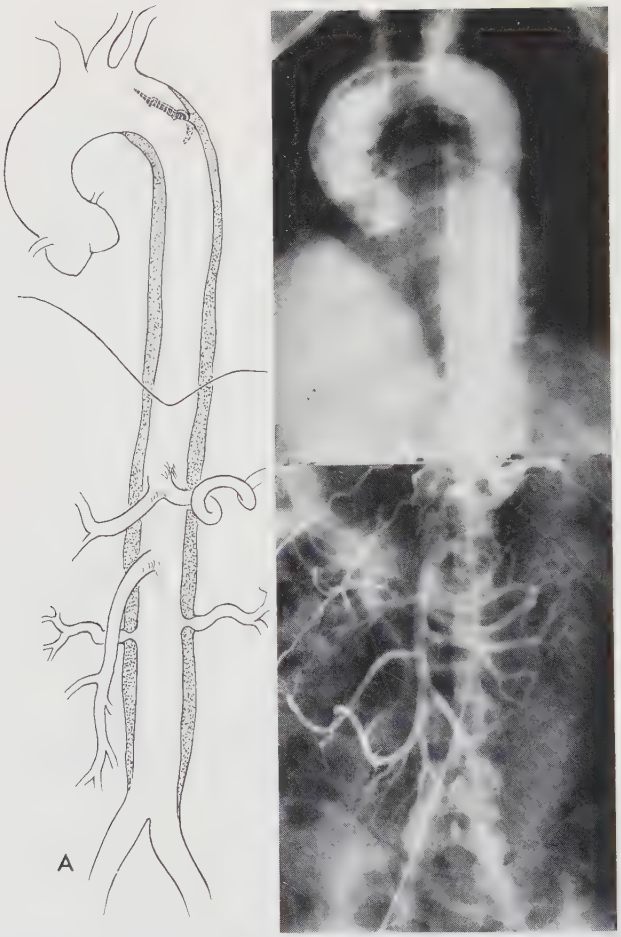


Figure 11. A patient with acute dissecting aortic aneurysm experienced pain, renal artery compression, and azotemia. *A*, An aortogram made before the operation showed the point of origin and extent of the dissection with poor visualization of the renal arteries.

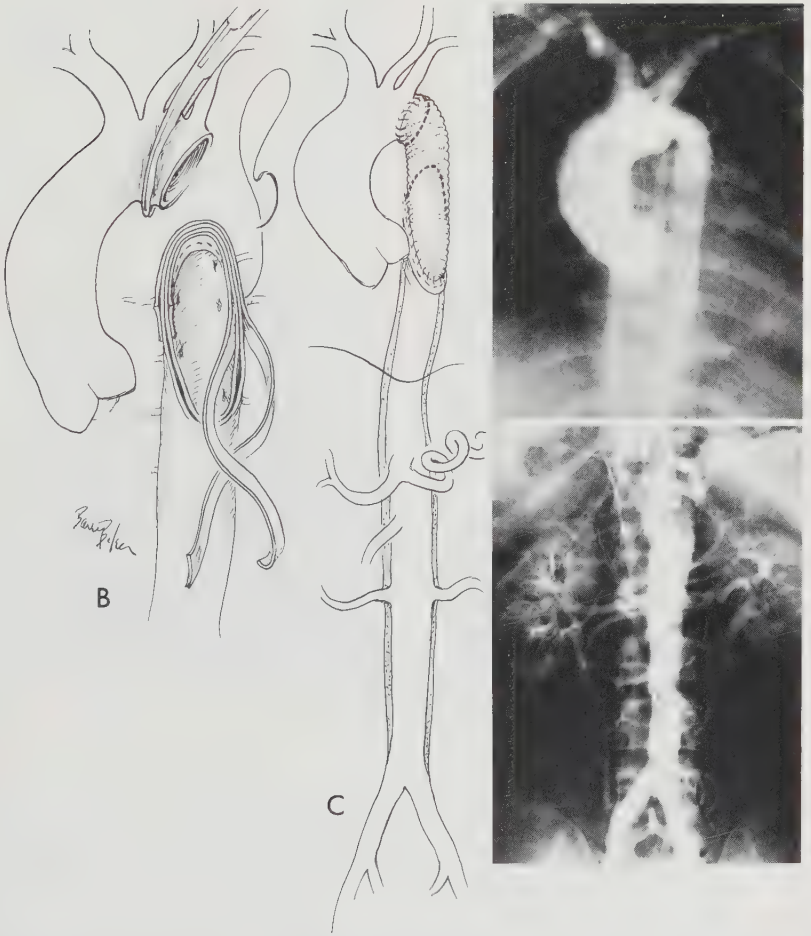


Figure 11 (*Continued*). *B,* During the operation only the point of origin was removed, sparing the intercostal arteries, and the distal aorta was reconstructed. *C,* An aortogram made after the operation showed the graft in place, with minimal signs of distal dissection, and patent vessels including the renal arteries.

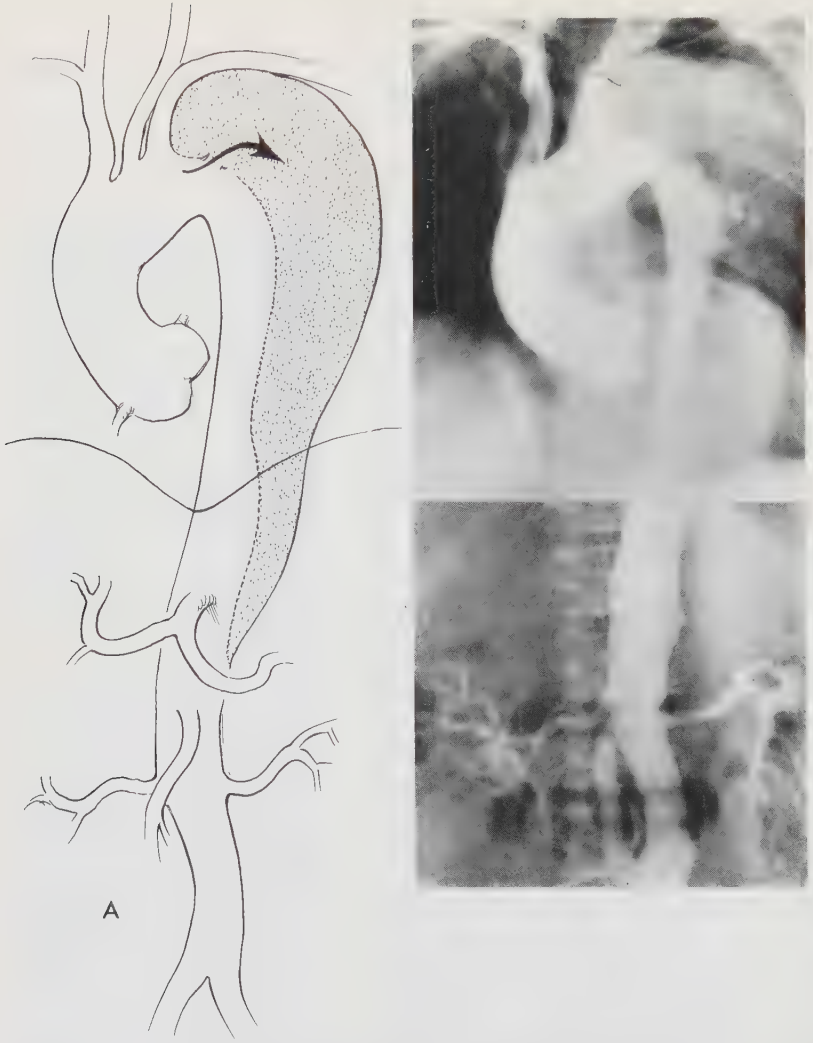


Figure 12. A fusiform dilatation of the thoracic segment was the result of a dissecting aortic aneurysm. *A*, An aortogram made before the operation showed the extent of the dissection.

patients with renal, mesenteric, and iliac artery obstruction. Abdominal operation is then rarely necessary and only when the proximal operation has failed to relieve distal obstruction.

The method of treating acute or chronic dilated dissections is shown in Figure 12. The dilated segment of disease, including the point of origin at the left subclavian artery, is replaced by tubular graft. Proximally, the tube is attached end-to-end to uninvolved aorta and distally to the bevelled reconstituted aorta to spare intercostal arteries. These operations have been employed in 35 patients, 18 with acute dissections and

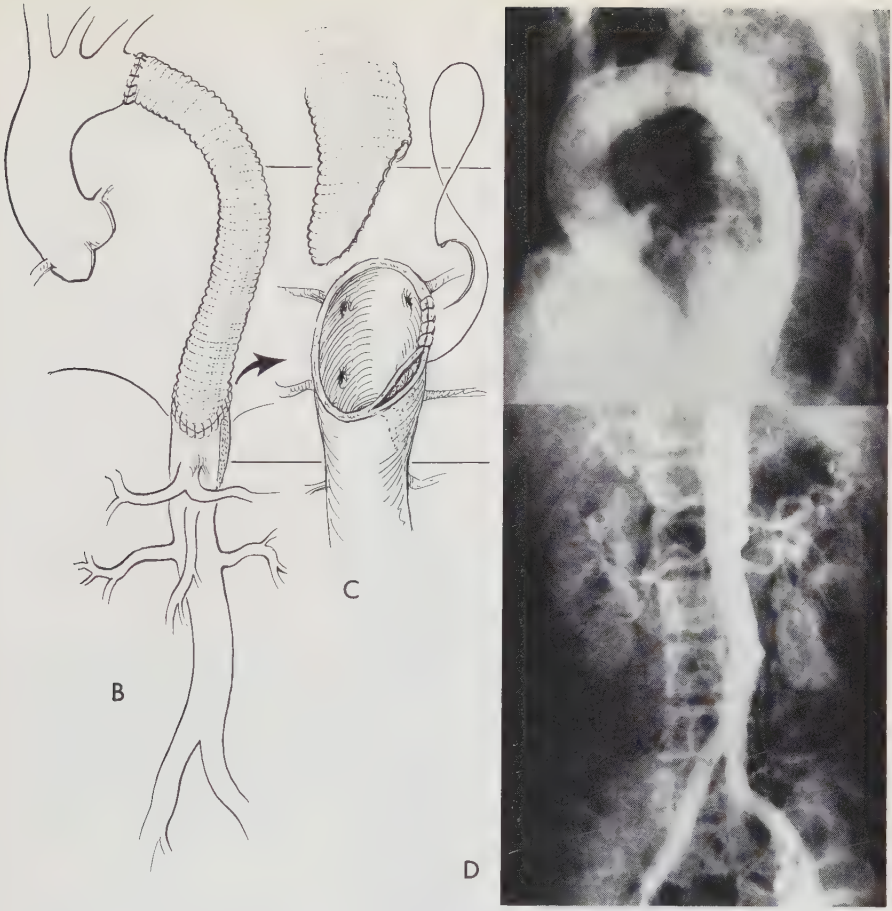


Figure 12 (*Continued*). *B* and *C*, During repair, the intercostal arteries were spared. *D*, A postoperative aortogram showed the graft in place and functioning.

17 with chronic dissections during the past eight years. Treatment was successful in 16 (89 percent) of the former and all of the latter patients.

Chronic Fusiform and Sacciform Aneurysms

Treatment in patients with chronic fusiform aneurysms is tubular graft replacement, and in the rare patient with sacciform aneurysms, insertion of a patch (Fig. 13). Tubular grafts are employed in fusiform aneurysms since the entire circumference is involved (Fig. 14). The preferred technique of inserting tubular grafts is by the inclusion technique (Fig. 15).[21] The advantages of this method are that minimal dissection is required for exposure, minimizing injury to adjacent structures, that the intercostal and mediastinal collateral circulation are preserved, and that the operative site is both tamponaded and excluded from adjacent structures by suture of the aneurysmal wall around the graft. Bypass shunts are not employed.[12]

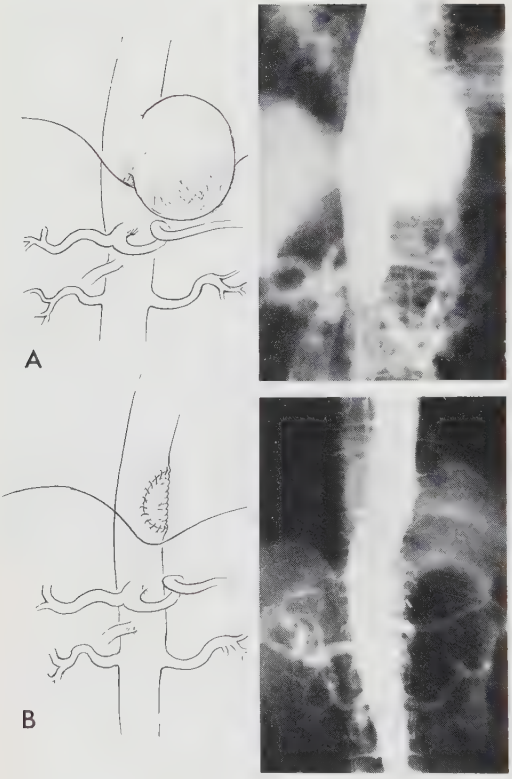


Figure 13. A sacciform aneurysm of the descending thoracic aorta was treated with a patch. *A*, A preoperative aortogram showed the location and size of the aneurysm. *B*, After operation the patch provided satisfactory aortic repair.

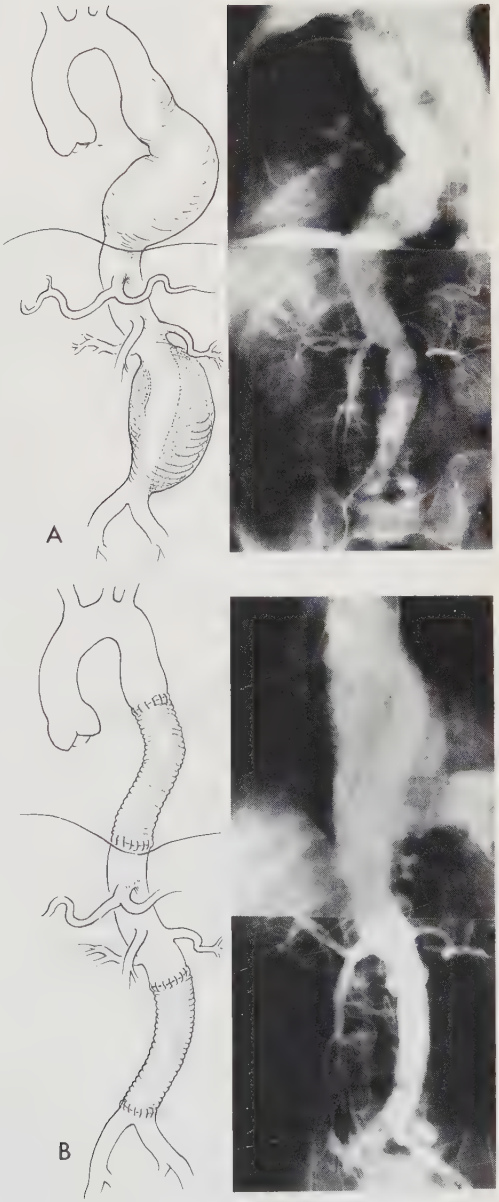


Figure 14. A 75-year-old man suffered symptomatic fusiform aneurysm of both the descending thoracic and abdominal aortas. *A*, Before operation, aneurysmal disease was extensive. *B*, Two grafts placed during operation functioned well postoperatively.

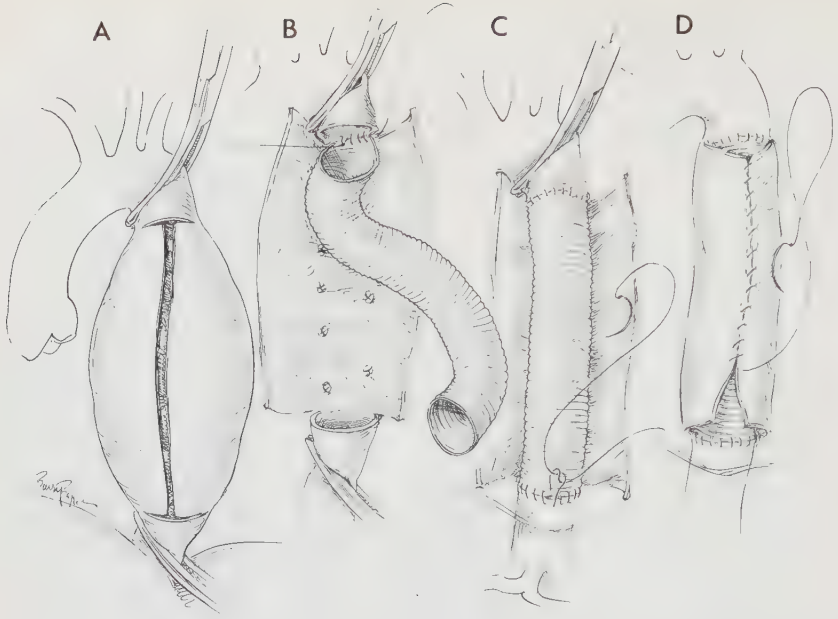


Figure 15. The technique of treating aneurysms of the descending thoracic aorta is shown. *A*, With minimal dissection, the aorta is exposed proximally and distally, clamped, and opened with cautery. *B* and *C*, Bleeding intercostal arteries are sutured and the graft is inserted. *D*, The aneurysmal wall is sutured around the graft.

The results of operation in these cases have been dependent upon a number of factors including time of treatment, whether or not a shunt has been used, and experience (Tables 1 and 2). Early in the experience using shunts, mortality was 25 per cent and the incidence of paraplegia was 9 per cent. With more extensive experience using better techniques and advanced supportive measures, mortality has been reduced to 7 per cent and paraplegia has not occurred in the last 59 patients treated. Most surgeons disagree with this approach and employ some type of shunt. They report a low but significant incidence of paraplegia. The fact of the matter is that paraplegia is the result of a number of factors not well understood. Consequently, this complication continues to occur regardless of technique employed at the present time.[5, 9, 10, 17, 25, 38]

Table 1. *Results According to Use of Shunts in Aneurysms of the Descending Thoracic Aorta*

	NUMBER OF CASES	PARAPLEGIA		SURVIVAL	
		Number	Per Cent	Number	Per Cent
Shunt	37	2	5	29	78
No shunt	86	2	2	76	88
TOTAL	123	4	3	105	85

Table 2. *Results According to Period of Operation in Aneurysms of the Descending Thoracic Aorta*

PERIOD	NUMBER OF PATIENTS	SURVIVAL		PARAPLEGIA	
		Number	Per Cent	Number	Per Cent
1st (7 years)	23	17	74	2	9
2nd (7 years)	41	33	80	2	5
3rd (8 years)	59	55	93	0	
TOTAL	123	105	85	4	3

THORACO-ABDOMINAL AND ABDOMINAL AORTIC ANEURYSMS

Thoraco-abdominal and abdominal aortic aneurysms involving the celiac, superior mesenteric, and renal arteries are relatively rare and pose special problems of aortic and arterial reconstruction. Both major body cavities may need to be entered, long segments of both the thoracic and the abdominal aorta may need to be replaced, and circulation must be restored to both the abdominal viscera and the lower extremities. The spinal cord blood supply must be restored in patients with extensive replacement of both the thoracic and the abdominal aortas.

Various techniques have been employed successfully in the treatment of thoraco-abdominal and abdominal aortic aneurysms. The original most popular method was insertion of a permanent end-to-side bypass graft of Dacron, with sidearm grafts which were attached sequentially to the visceral arteries beginning with the left renal artery, in order to minimize renal ischemic time during resection of aneurysms. These procedures proved difficult and lengthy, and were associated with considerable blood loss and a mortality rate of 26 per cent. We prefer the graft inclusion technique for aneurysms of the descending thoracic aorta (Fig. 16). The graft is simply inserted inside the aneurysm and the visceral arteries are reattached to openings made in the graft. The technique in this position is particularly advantageous, since time-consuming dissection leading to bleeding and damage to adjacent structures is avoided, and the need for sidearm grafts with their multiple anastomoses and their tendency to kink and obstruct is eliminated.

Exposure is obtained by thoraco-abdominal incision. The thoracic aorta is exposed by retracting the lung, which is temporarily collapsed by Carlen's double lumen tube, and the abdominal aorta is exposed retroperitoneally by mobilizing the left colon, the kidney, the spleen, the tail of the pancreas, and the stomach by incising the peritoneum along the left gutter. These structures are retracted upward and to the right. The aorta is clamped proximally and distally and without the use of shunts; the aneurysm is entered through a longitudinal incision made by cautery. The edges of the aneurysmal wall are retracted by stay sutures. The graft is attached proximally, the visceral vessels are attached to one or more openings made in the graft, and the graft is flushed

Figure 16. *A*, A thoraco-abdominal aortic aneurysm involving the distal thoracic aorta and the abdominal aorta proximal to a previously inserted Dacron bifurcation graft appeared in this patient.

and clamped beyond the visceral arteries. The proximal clamp is then removed, restoring visceral arterial flow, and the reconstruction is completed by performing the distal anastomosis (see Fig. 16, *B* and *C*).

This technique is adaptable to all extensive aneurysms in the thoraco-abdominal area with certain modifications (Figs. 17 to 20). Replacement of extensive aneurysms involving long segments of both thoracic and abdominal aortas may be associated with spinal cord ischemia from extensive interruption of intercostal and lumbar arteries. These vessels are reattached in these cases by the techniques shown in Figures 17 and 19.

Figures 18 and 19 show patients with diffuse enlarging chronic dissections. The dissected dilated circumference of the descending thoracic aorta is replaced with a part of a tube graft, preserving the undissected circumference from which the intercostal and upper lumbar arteries arise, in order to maintain spinal cord circulation. The undissected circumference in patients with chronic aneurysm is firm, fibrous, and holds sutures well without intimal tearing. In one acute case, the undissected segment was friable, did not hold sutures well, and the so-called normal intima was cut by sutures in several places, permitting the dissection to recur. The procedure is ideal in patients with chronic dissections, but may have limited application in patients with acute dissection. Eleven chronic dissections and one acute dissection involving the descending thoracic and abdominal aorta were treated in this series.

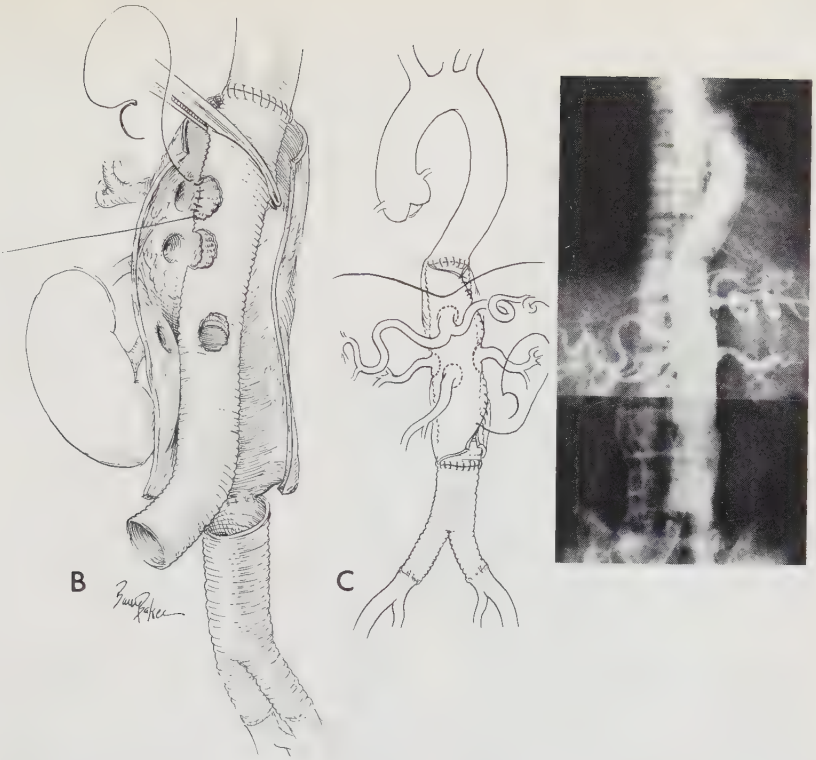


Figure 16 (*Continued*). *B*, The patient was treated with an additional graft. *C*, Postoperatively, the graft was in place and blood flow was seen in all reattached arteries.

All of the former patients are long-term survivors with well controlled disease. Most of these had had previous resections of both the ascending aorta and varying segments of the descending thoracic aorta (Fig. 19). Thoraco-abdominal operation was performed for progressive enlargment of the distal false lumen or for recurrent pain of dissection. These cases emphasize the effectiveness of operation in the chronic form and the need for long-term follow-up in the patient with arrested acute dissection because of the progressive nature of the disease process in some patients.

Aneurysms involving the entire abdominal aorta are treated as described above for more extensive lesions (Fig. 20). Infrarenal aortic aneurysms that involve the renal arteries, however, are exposed by the same method commonly employed in the treatment of infrarenal aortic aneurysm. The principal differences are that exposure is required up to the superior mesenteric artery, and that the aorta is cross clamped in the aortic hiatus of the diaphragm for proximal control. The graft is inserted in the usual fashion and the renal arteries are reattached either by direct anastomosis to openings made in the graft or by secondary grafts attached proximally to the aortic graft and distally to the ends of the renal arteries.

Figure 17. An extensive fusiform aneurysm in the descending thoracic and upper abdominal aorta was treated by the technique of graft inclusion and direct vessel reattachment. *A*, An aortogram made before the operation showed extensive disease.

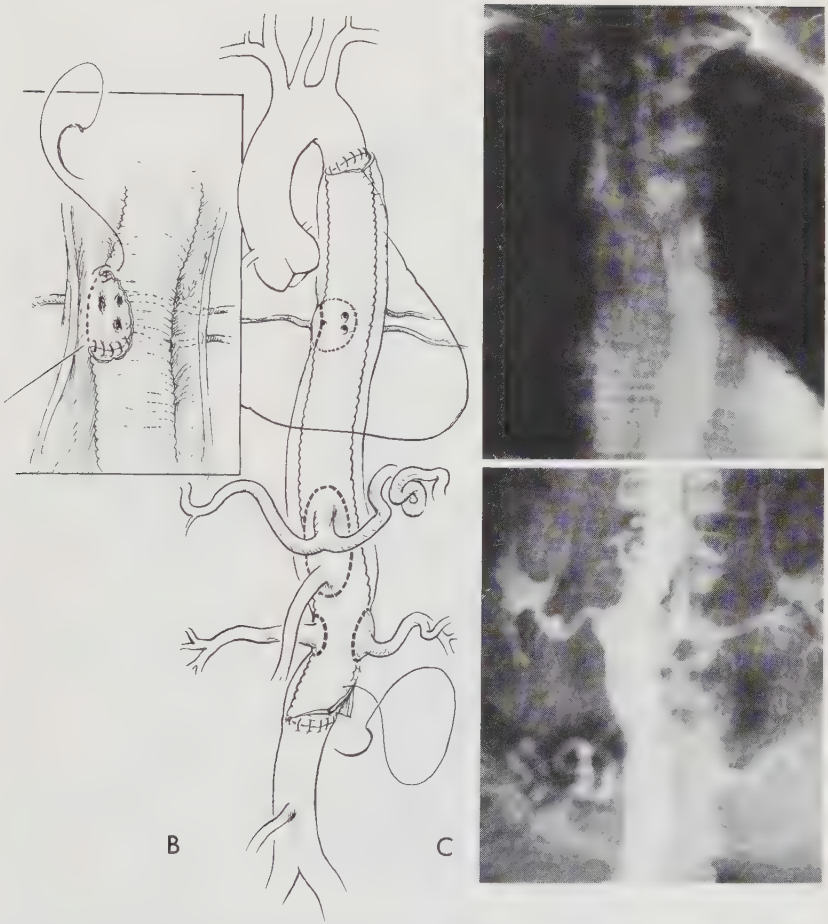


Figure 17 (*Continued*). *B*, In extensive disease, the intercostal arteries are reattached to prevent paraplegia. *C*, An aortogram made after the operation showed the graft in place and the patent reattached arteries, including the intercostal artery.

Figure 18. Preoperative aortography showed a chronic recurrent dissecting aneurysm of the descending thoracic and abdominal aorta.

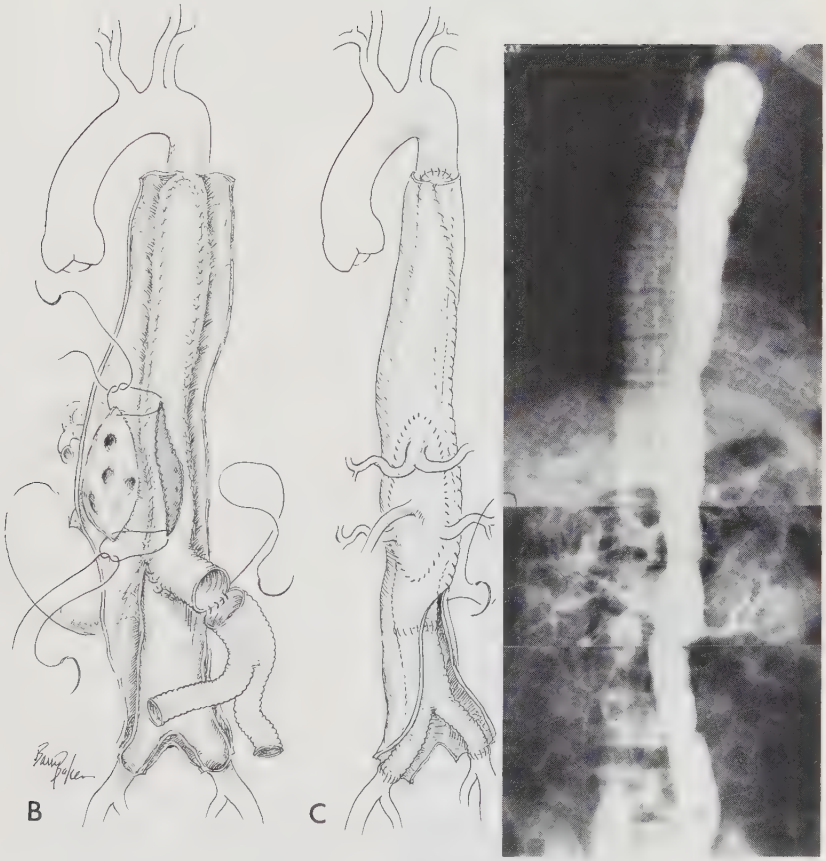


Figure 18 (*Continued*). *B*, A graft was placed and the visceral vessels were reattached. *C*, An aortogram made after the operation showed the reconstructed aorta, preserved intercostal arteries, and reattached visceral arteries.

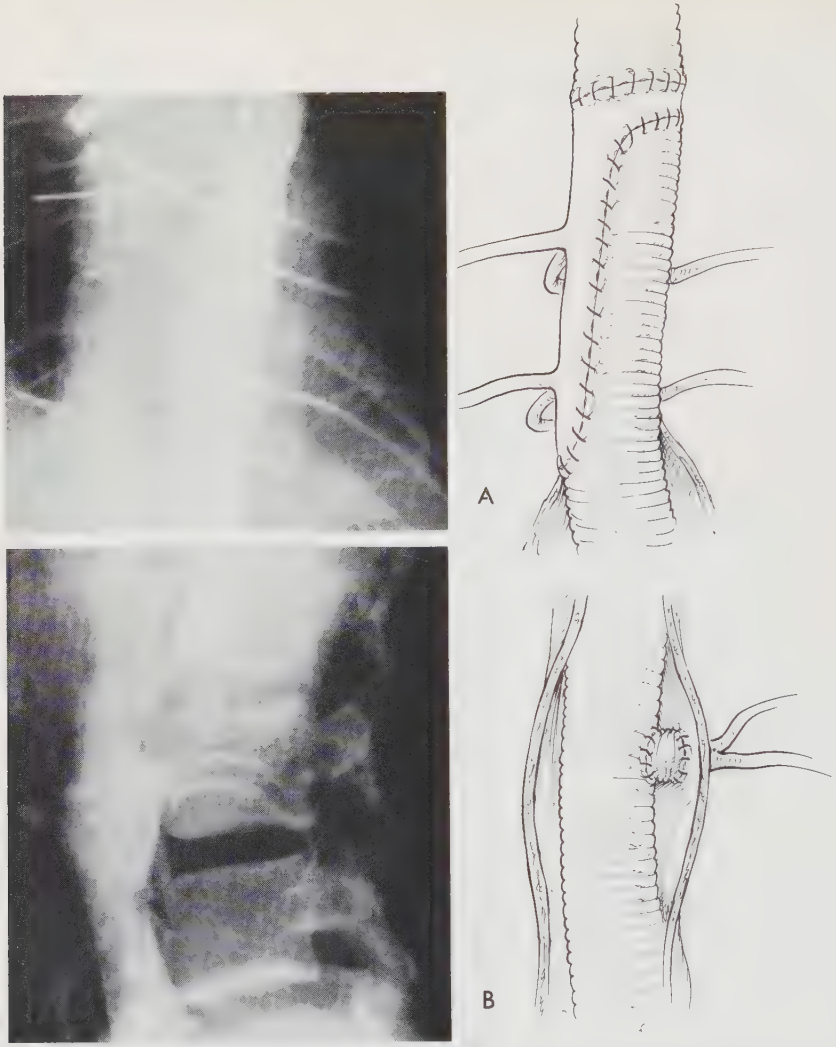


Figure 19. A patient suffered an extensive chronic recurrent dissecting aortic aneurysm in the lower descending thoracic and abdominal aorta. The ascending and proximal descending thoracic aorta had been removed previously at separate operations. *A*, During operation, the intercostal arteries were preserved. *B*, Using a graft, the lumbar artery was reattached.

The extent of involvement of the aorta by aneurysm and the operative results in 99 patients in this series of extensive cases are shown in Table 3. Of the 99 patients treated, 90 (91 per cent) survived, five with lower extremity weakness (three who died also had neurologic deficits). One of these completely recovered; three were rehabilitated; and another recent case is still disabled. Most of these complications occurred in patients with involvement of extensive segments of both the thoracic and abdominal aorta. Of the 27 patients in this group, six (22 per cent)

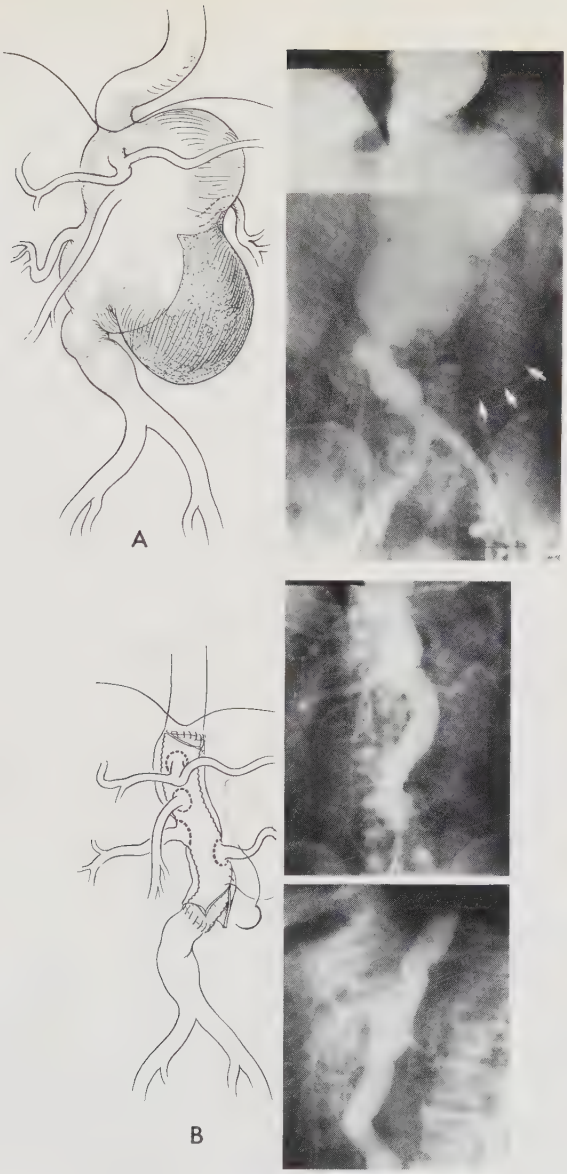


Figure 20. *A*, A preoperative aortogram revealed an aneurysm involving the proximal abdominal aorta. *B*, Postoperatively, the directly reattached visceral vessels were functioning well.

suffered some form of neurologic deficit (Tables 3 and 4). Of these, 19 had successful reattachment of intercostal or lumbar arteries or both and only three (15 per cent) suffered deficits, whereas in eight patients in whom this was not accomplished, three suffered deficits, suggesting that reattachment of intercostal and lumbar arteries is helpful in preventing paraplegia.

Table 3. *Results in Thoraco-Abdominal and Abdominal Aortic Aneurysms Involving the Celiac, Superior Mesenteric, and Renal Arteries*

Group	Involvement	Number of Patients	Paraplegia		Survival	
			Number	Per Cent	Number	Per Cent
I	Thoracic and abdominal aorta without vessels	12	1	8	12	100
II	Entire aorta	1	0		0	
III	Most of the descending thoracic abdominal aorta with vessels	27	6	22	23	85
IV	Distal thoracic and abdominal aorta with vessels	23	1	4	20	87
V	Abdominal aorta with vessels	21	0		20	95
VI	Abdominal aorta with renal arteries	15	0		15	100
	TOTAL	99	8	8	90	91

Table 4. *Correlation of Paraplegia with Preservation of Intercostal and Lumbar Arteries in 27 Patients with Extensive Involvement of the Thoracic and Abdominal Aortas*

RESULTS OF REATTACHMENT OF INTERCOSTAL AND LUMBAR ARTERIES	NUMBER OF PATIENTS	PARAPLEGIA				
		Number	Per Cent	Mild	Moderate	Severe
Successful	19	3	16	2	1	0
Not accomplished	8	3	38	0	1	2
Not successful	(3)		(1)	(0)	(0)	(1)
No attempt	(5)		(2)	(0)	(1)	(1)
TOTAL	27	6	22	2	2	2

Table 5. *Renal Function after Operation Correlated with Renal Artery Occlusion Time (in 95 Patients Leaving Operating Room)**

DURATION OF OCCLUSION (MINUTES)	NUMBER OF PATIENTS	RENAL DYSFUNCTION		DIALYSIS
		Number	Per Cent	
15 to 35	35	4	11	1
35 to 55	50	1	2	0
55 to 150	8	3	38	3
TOTAL	93	8	9	4**

*One patient was on chronic hemodialysis
**Death occurred in two patients

Renal function was carefully observed in these patients and correlated with the period of renal ischemia during operation, which varied from 15 to 150 minutes (Table 5). Mild transient elevations of creatinine occurred in four patients, and acute tubular necrosis developed in four requiring hemodialysis. Most of the latter endured long ischemic times as a result of repetition of renal artery reconstruction when aortography showed occluded renal arteries. Two patients in the last group survived, and all of the first group recovered and returned to preoperative levels of renal function.

Infrarenal Aortic Aneurysms

Mortality and morbidity of operation in patients with infrarenal abdominal aortic aneurysm have steadily declined during the past 15 years, and now varies between three and five per cent in elective cases.[16, 33] The overall mortality in 687 patients in our series treated during the past 23 years was 6.7 per cent and varied according to the presence of rupture. The survival rate in 631 (92 per cent) of unruptured cases was 96 per cent and 71.4 per cent in 56 (8 per cent) with rupture. Progress in our own experience is evident by the fact that mortality among the last 329 patients was 3 per cent (10 patients), in spite of associated cardiopulmonary and other atherosclerotic diseases such as unsuspected stroke disease, coronary artery disease, hypertension, peripheral vascular disease, and obesity in many patients. Many of the patients are elderly; however, age itself does not produce excessive risk since a mortality of only 4.7 per cent has been reported in patients over 80 years of age who were submitted to elective operation.[28]

Graft Inclusion Technique of Operation

The most important technical advance in graft replacement of abdominal aortic aneurysms was the introduction of graft inclusion technique by Javid and associates which the authors and others subsequently adopted both for thoracic and abdominal aortic aneurysms.[21] The tremendous advantages in its application have been described above.

Many surgeons rejected the technique, since bacteria could be cultured from the aneurysmal contents in some cases, and insisted on the original technique of excision and graft replacement. The signifi-

cance of positive bacterial culture is unknown since infection of the graft site after this operation is rare and probably may be cured by prophylactic antibiotics.[37] Should nonsusceptible organisms be cultured, appropriate antibiotics should be added. We would not culture unless infection was obvious. Originally, we endarterectomized the aneurysmal wall to produce a fresh healing surface that would stick to and heal solidly around the graft. This has been abandoned and in the last 185 patients the clot has simply been removed and the aneurysmal wall sutured around the graft. This has simplified the procedure, and complications including the dreaded graft infection and duodenal erosion have not occurred.

The methods of application are shown in Figures 21 to 23 and are dependent upon associated common iliac artery disease. The aneurysm in 61 per cent of cases was confined to the infrarenal segment of the abdominal aorta, and the distal iliac arteries were not involved either by aneurysm or occlusive disease. In these patients, the distal aorta was simply replaced with a straight woven Dacron tube (Fig. 21). The iliac arteries were involved by disease in the remaining patients and a bifurcation graft was required (Figs. 22 and 23). The iliac limbs of the graft were attached end-to-end to the distal common iliac arteries in patients with proximal common iliac artery involvement and end-to-side to either the external iliac or common femoral artery when extensive segments were involved.

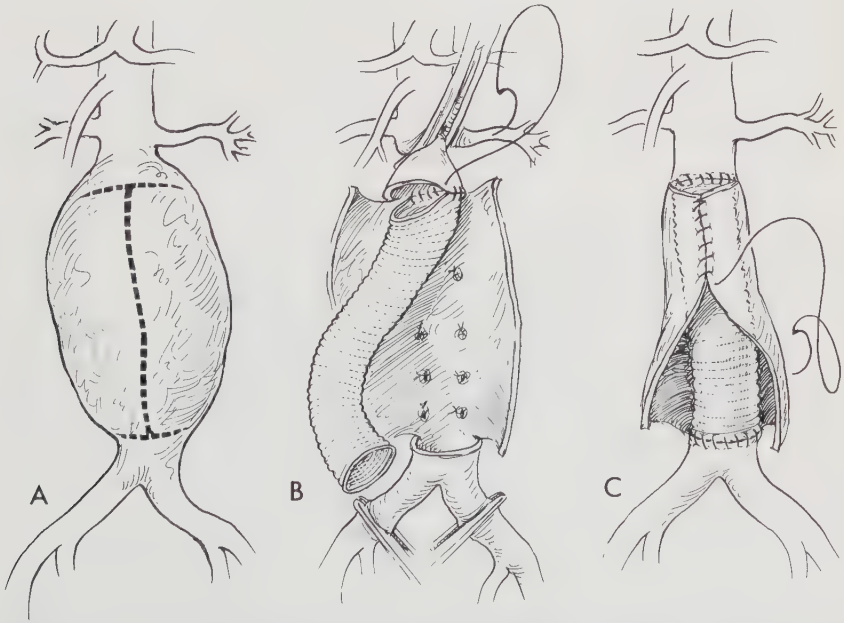


Figure 21. The graft inclusion method of treatment of infrarenal abdominal aortic aneurysms not involving the iliac arteries is shown. *A*, Incisions are made in the aneurysm. *B*, the lumbar arteries are sutured and graft anastomosis is made by continuous sutures. *C*, The operation is completed by suturing the aneurysmal wall and the retroperitoneal tissues around the graft.

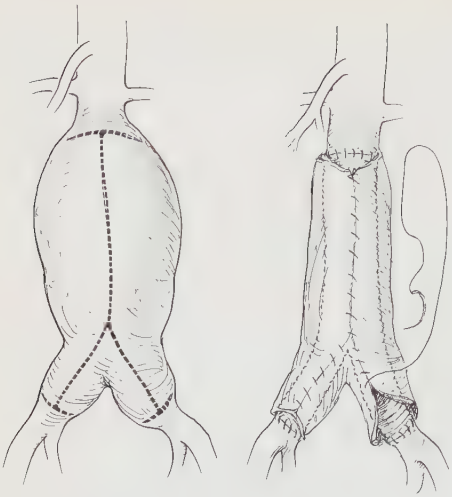


Figure 22. In patients with involvement of the proximal common iliac artery, abdominal aortic aneurysms are replaced by grafts as shown.

Ruptured Abdominal Aortic Aneurysms

Operative treatment of the patient with ruptured abdominal aortic aneurysm must not be delayed because resuscitation can be accomplished only after the aorta has been clamped proximally. The patient with hypotension must have blood drawn for saline and routine methods

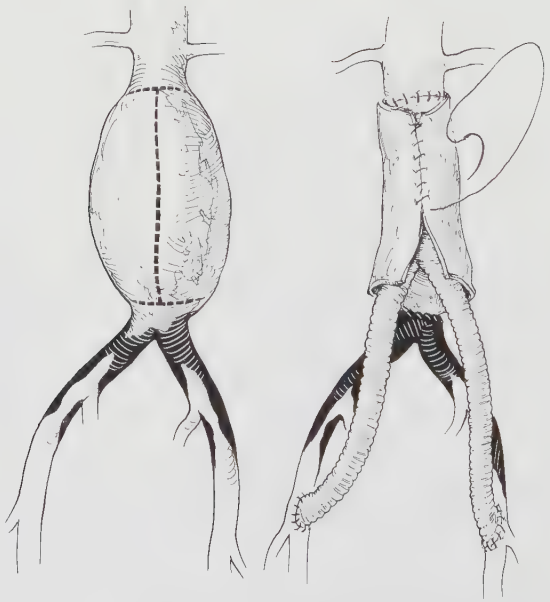


Figure 23. The graft inclusion method is shown as it is used in the treatment of abdominal aortic aneurysms in the presence of associated disease of the common and external iliac arteries.

of typing and cross-matching. Intravenous bilateral subclavian lines must be connected with large bore catheters. The patient should be taken directly to the operating room. Minimal amounts of fluids or vasopressors should be given to avoid a rise in the blood pressure which would cause bleeding to recur or increase. If time permits, the patient may be washed and draped. He is then anesthetized, the abdomen opened, and the aorta compressed manually against the lumbar spine at the level of aortic hiatus in the diaphragm. Blood, colloid, and electrolyte solutions, as described above, should be given rapidly until normal pressure and cardiac action are restored as determined by clinical estimation. A radial arterial line is then inserted for monitoring of the blood pressure by the anesthetist, and the aorta is exposed by blunt dissection and clamped at the aortic hiatus. The aneurysm is opened, the proximal anastomosis is made, and the clamp is placed across the infrarenal segment of graft, restoring blood flow to the abdominal viscera. Sodium bicarbonate and mannitol solution should be given, the latter as previously described. At this point, the various monitoring devices are added as the surgeon completes the distal anastomosis, and the detected abnormalities are corrected, including raising the central venous pressure to normal levels with blood, colloid, and Ringer's lactate solution. The aneurysmal wall is then sutured around the graft, but no dissection is performed in the retroperitoneal hematoma.

Similar patients with normal vital signs should be taken to the preinduction room or intensive care unit outside the operating room. The standard preparation should be carried out rapidly before operation is started. Traditional studies such as blood, urine, electrocardiograms, roentgenograms, and others should be omitted, because these delay treatment and do not alter surgical treatment in these desperate cases. After completion of the operation, all the necessary studies of associated disease, blood gases, electrolytes, and renal function should be completed and the necessary corrections made.

Spontaneous Aorto-Caval Fistula

Abdominal aortic aneurysms may rupture spontaneously into the vena cava or the iliac veins, and produce aorto-caval fistula. The characteristic symptom is sudden development of edema of the body and legs with heart failure. The abnormal findings include a pulsating abdominal mass with a murmur typical of aorto-caval fistula and prominent superficial veins over the surface of the abdomen. Treatment employs diuretics, cardiac medications, and early operation. The technique of operation successfully employed in five patients in our series was essentially the same as that used in routine elective operation for aneurysm. Vena caval bleeding was controlled by pressure over the vena caval opening with the left hand after opening of the aneurysm, and the defect in the aneurysmal wall was closed with interrupted sutures. Blood loss was minimal and vena caval replacement was not necessary in these cases. All five of our patients survived, and the systemic effects of arteriovenous fistula were rapidly reversed.

Aorto-Duodenal Fistula

Abdominal aortic aneurysms may erode and rupture into the fourth part of the duodenum or the first part of the jejunum. The salient features of this disorder are the presence of a pulsating abdominal mass, accompanied by gastrointestinal bleeding. The latter is frequently intermittent and mild to moderate, but eventually becomes massive. Treatment is immediate operation using the graft inclusion technique. The opening in the aneurysm is closed by suture after the graft is inserted, and the small intestine is either closed by suture or the involved segment is resected. In any case the outer aneurysmal wall is sutured tightly around the graft and separated from the gastrointestinal tract by omentum. The four patients in our series survived operation with good long-term results. Infection and recurrent fistula did not occur. Antibiotics were given only during the period of hospitalization.

Other Considerations

Space limitations do not permit discussion of other interesting facets of abdominal aortic aneurysm including associated horseshoe kidney, aberrant renal arteries, retroperitoneal fibrosis with ureteral obstruction, and occlusive or aneurysmal disease of aortic branches. Mycotic aneurysms, traumatic aneurysms with and without venous communication, presentations of patients unsuccessfully treated by various methods of wrapping and wiring, abdominal dissections, aneurysm and pregnancy, peripheral emboli, presentation of aneurysm by manifestions of disseminated intravascular coagulopathy, and long-term complications such as anastomotic aneurysms and graft obstruction are other subjects of discussion. The interested reader is referred to the literature.

ACKNOWLEDGMENT

Grateful acknowledgment is given for illustrations to Mr. Barry Baker, Department of Medical Illustration and Audiovisual Education, Baylor College of Medicine, Houston, Texas.

REFERENCES

1. Appelbaum, A., Karp, R. B., and Kirklin, J. W.: Ascending vs. descending aortic dissections. Ann. Surg., *183*:296, 1976.
2. Attar, S., Fardin, R., Ayella, R., et al.: Medical vs. surgical treatment of acute dissecting aneurysms. Arch. Surg., *103*:568, 1971.
3. Bentall, H., and DeBono, A.: A technique for complete replacement of the ascending aorta. Thorax, 23:338, 1968.
4. Bernstein, E. F., Dilley, R. B., Goldberger, L. F., et al.: Growth rates of small abdominal aortic aneurysms. Surgery, *80*:765, 1976.
5. Bloodwell, R. D., Hallman, G. L., and Cooley, D. A.: Partial cardiopulmonary bypass for pericardiectomy and resection of descending thoracic aortic aneurysms. Ann. Thorac. Surg., 6:46, 1968.

6. Bloodwell, R. D., Hallman, G. L., and Cooley, D. A.: Total replacement of the aortic arch and the "subclavian steal" phenomenon. Ann. Thorac. Surg., 5:236, 1968.
7. Brewster, D. C., Darling, R. C., Raines, J. K., et al.: Assessment of abdominal aortic aneurysm size. Circulation, 56:164, 1972.
8. Brewster, D. C., Retana, A., Waltman, A. C., et al.: Angiography in the management of aneurysms of the abdominal aorta. Its value and safety. N. Engl. J. Med., 292:822, 1975.
9. Connolly, J. E., Wakabayashi, A., German, J. C., et al.: Clinical experience with pulsatile left heart bypass without anticoagulation for thoracic aneurysms. J. Thorac. Cardiovasc. Surg., 62:568, 1971.
10. Connors, J. P., Ferguson, T. B., Weldon, C. S., et al.: The use of the TDMAC-heparin shunt in replacement of the descending thoracic aorta. Ann. Surg., *181*:735, 1975.
11. Crawford, E. S., Morris, G. C., Jr., Howell, J. F., et al.: Operative risk in patients with previous coronary artery bypass. Ann. Thorac. Surg., 26:215, 1978.
12. Crawford, E. S., and Rubio, P. A.: Reappraisal of adjuncts to avoid ischemia in the treatment of aneurysms of descending thoracic aorta. J. Thorac. Cardiovasc. Surg., 66:693, 1973.
13. Crawford, E. S., Snyder, D. M., Cho, G. C., et al.: Progress in treatment of thoraco-abdominal and abdominal aortic aneurysms involving celiac, superior mesenteric, and renal arteries. Ann. Surg., *188*:404, 1978.
14. Darling, R. C., Messina, C. R., R. N., Brewster, et al.: Autopsy study of unoperated abdominal aortic aneurysms. Circulation, 56:161, 1977.
15. DeBakey, M. E., Crawford, E. S., Cooley, D. A., et al.: Successful resection of fusiform aneurysm of aortic arch with replacement by homograft. Surg. Gynecol. Obstet., *105*: 657, 1957.
16. DeBakey, M. E., Crawford, E. S., Cooley, D. A., et al.: Aneurysm of abdominal aorta: Analysis of results of graft replacement therapy one to eleven years after operation. Ann. Surg., *160*:622, 1964.
17. Donahoo, J. S., Brawley, R. K., and Gott, V. L.: Heparin-coated vascular shunt for thoracic aortic and great vessel procedures: A 10-year experience. Ann. Thorac. Surg., 23:507, 1977.
18. Edwards, S. W., and Kerr, A. R.: A safer technique for replacement of the entire ascending aorta and aortic valve. J. Thorac. Cardiovasc. Surg., 59:837, 1970.
19. Griepp, R. B., Stinson, E. B., Hollingsworth, J. F., et al.: Prosthetic replacement of the aortic arch. J. Thorac. Cardiovasc. Surg., 70:1051, 1975.
20. Hufnagel, C. A., and Conrad, P. W.: Dissecting aneurysms of the ascending aorta: direct approach to repair. Surgery, *51*:84, 1962.
21. Javid, H., Hulian, O. C., Dye, W. S., et al.: Complications of abdominal aortic grafts. Arch. Surg., 85:650, 1962.
22. Kaiser, A. B., Clayson, K. R., Mulherin, J. L., et al.: Antibiotic prophylaxis in vascular surgery. Ann. Surg., *188*:283, 1978.
23. Kidd, J. N., Ruel, G. J., Jr., Cooley, D. A., et al.: Surgical treatment of aneurysms of the ascending aorta. Circulation., 54:118, 1976.
24. Kouchoukos, N. T., Karp, R. B., and Lell, W. A.: Replacement of the ascending aorta and aortic valve with a composite graft: Results in 25 patients. Ann. Thorac. Surg., 24:140, 1977.
25. Lawrence, G. H., Hessel, E. A., Sauvage, L. R., et al.: Results of use of the TDMAC-Heparin shunt in surgery of aneurysms of descending thoracic aorta. J. Thorac. Cardiovasc. Surg., 73:393, 1977.
26. Liddicoat, J. E., Bekassy, S. M., Rubio, P. A., et al.: Ascending aortic aneurysms: Review of 100 consecutive cases. Circulation., *51–52*:1, 1974.
27. Najafi, H., Dye, W. S., Javid, H., et al.: Aortic insufficiency secondary to aortic root aneurysm on dissection. Arch. Surg., *110*:1401, 1975.
28. O'Donnell, T. F., Jr., Darling, R. C., and Linton, R. R.: Is 80 years too old for aneurysmectomy? Arch. Surg., *111*:1250, 1976.
29. Ott, D. A., Frazier, O. H., and Cooley, D. A.: Resection of the aortic arch using deep hypothermia and temporary circulatory arrest. Circulation., 58:227, 1978.
30. Reul, G. J., Jr., Cooley, D. A., Hallman, G. L., et al.: Dissecting aneurysms of the descending aorta: Improved surgical results in 91 patients. Arch. Surg., *110*:632, 1975.
31. Shawker, T. H., and Steinfeld, A. D.: Ultrasonic evaluation of pulsatile abdominal masses. J.A.M.A., 239:419, 1978.
32. Stephens, D. H., Williamson, B., Jr., Sheedy, P. F., II, et al.: Computed tomography of the retroperitoneal space. Radiol. Clin. North Am., *15*:377, 1977.
33. Thompson, J. E., Hollier, L. H., Patman, R. D., et al.: Surgical management of abdominal aortic aneurysms: Factors influencing mortality and morbidity – A 20-Year experience. Ann. Surg., *181*:654, 1975.
34. Trinkle, J. K., Discussion, Najafi, et al.: Aortic insufficiency secondary to aortic root aneurysm on dissection. Arch. Surg., *110*:1401, 1975.

35. Wheat, M. W., Jr., Palmer, R. F., Bartley, T. D., et al.: Treatment of dissecting aneurysms of the aorta without surgery. J. Thorac. Cardiovasc. Surg., 50:364, 1965.
36. Wheat, M. W., Jr., and Palmer, R. F.: Dissecting aneurysms of the aorta. Curr. Probl. Surg., 1971.
37. Williams, R. D., and Fisher, F. W.: Aneurysm contents as a source of graft infection. Arch. Surg., *112*:415, 1977.
38. Wolfe, W. G., Kleinman, L. H., Weckler, A. S., et al.: Heparin-coated shunts for lesions of the descending thoracic aorta. Arch. Surg., *112*:1481, 1977.
39. Wolfe, W. G., and Moran, J. F.: The evolution of medical and surgical management of acute aortic dissection (Editorial). Circulation., 56:503, 1977.

The Methodist Hospital
6516 Bertner Avenue
Houston, Texas 77030

Visceral Artery Syndromes and Reconstructions

Ronald J. Stoney, M.D., and Cornelius Olcott, IV, M.D.†*

Interest in the management of occlusive lesions involving the visceral vessels has increased tremendously in the last decade as a result of greater awareness of their clinical significance, improved angiographic techniques that allow more accurate delineation, and improved surgical techniques that have resulted in effective, durable revascularizations with acceptable rates of morbidity and mortality.

There are two major categories of syndromes of intestinal ischemia. Acute ischemic syndromes are a result of embolic occlusion of a visceral branch of the abdominal aorta, thrombotic occlusion of a diseased visceral artery, and nonocclusive mesenteric vascular insufficiency.[1, 2, 6, 7, 10] Chronic intestinal ischemia usually results from stenotic or occlusive atherosclerotic lesions involving one or more of the visceral vessels.[4, 9, 11, 12, 14] A second, though less common, cause of chronic ischemia is extrinsic compression of the celiac axis by the median arcuate ligament, an entity that remains controversial to this date.[5, 8, 13, 14]

CLINICAL PRESENTATION

Acute Ischemia

Every attempt should be made to differentiate between various causes of acute intestinal ischemia, because treatment and prognosis differ.

The most prominent finding, regardless of cause, is the acute onset of abdominal pain, usually crampy, epigastric, or periumbilical and characteristically described as out of proportion to the physical findings. The onset of nausea and vomiting, and, less commonly, diarrhea typically follows the pain. Gross intestinal bleeding is uncommon, though the stool may be positive for occult blood. Physical examination usually

*Associate Professor of Surgery, University of California, San Francisco, California
†Assistant Clinical Professor of Surgery, University of California, San Francisco, California

reveals mild diffuse tenderness associated with normal or slightly decreased bowel sounds. Frank peritoneal findings do not develop until late in the course of the disease. The leukocyte count is characteristically high, usually in excess of 20,000 per mm^3, although the patient is afebrile. Later, shock as a result of loss of vascular volume into ischemic bowel and superimposed sepsis appear.

Acute embolic occlusion typically occurs in patients with known heart disease, and frequently these patients will give a history of prior embolic events involving the lower extremity or cranial (cerebral) vessels. Acute thrombotic occlusion usually occurs in a vessel with preexisting atherosclerosis. These patients often give a history compatible with previous intestinal angina and frequently have sustained a significant weight loss. Thrombotic lesions tend to be more insidious in onset than embolic lesions. Nonocclusive arterial insufficiency is seen in conjunction with various forms of cardiac insufficiency, congestive heart failure, shock, aortic valve disease, use of vasopressors, and digitalis toxicity. Ischemia in these patients is primarily due to a defect in perfusion. However, associated stenotic lesions of the visceral vessels also have been described.[6, 10]

Arteriography is extremely helpful in differentiating the cause of acute intestinal ischemia. Embolic lesions characteristically involve the superior mesenteric artery and produce a defect several centimeters from the origin of the vessel. Acute thrombotic lesions involve the origin of the vessel and may involve one or more of the three visceral vessels. The angiographic appearance of nonocclusive ischemia is segmental arterial constriction of the mesenteric vessels, which may be associated with a proximal stenotic lesion, particularly involving the superior mesenteric artery.

Findings at exploration also help to differentiate thrombotic from embolic intestinal ischemia. Because an embolus tends to lodge several centimeters from the origin of the superior mesenteric artery, the proximal jejunum, which is supplied by the proximal superior mesenteric artery, is usually spared. With thrombotic occlusion of the superior mesenteric artery, the entire small bowel and proximal one-half of the colon are involved in the ischemic process.

The key to the diagnosis of acute intestinal ischemia is a high index of suspicion as these patients usually have a paucity of objective findings when first examined. Multiple recent reports have emphasized the importance of prompt evaluation in order to salvage as many patients as possible. This necessitates immediate angiography and exploration if the diagnosis is confirmed.[1, 2, 7]

Chronic Ischemia

The hallmark of chronic ischemia is chronic abdominal pain associated with involuntary weight loss. Changes in bowel evacuation pattern may also occur, though they are less common. The pain is typically epigastric, occurs as a dull ache or colic, and is postprandial. The pain characteristically comes on 15 to 30 minutes after eating and lasts for 1 to 3 hours before disappearing. The pain associated with eating becomes

so severe that patients develop a "food fear" and limit their oral intake predominantly to liquids, which results in the pronounced weight loss. Three-quarters of the patients are women, with a mean age of 57 years.

On examination, the patient is characteristically thin and may appear chronically ill, mimicking a patient with malignant disease of the viscera. Examination of the abdomen will not reveal acute findings. An epigastric bruit is frequently heard. Atherosclerotic involvement of other peripheral arteries is frequently noted. In cases of compression of the celiac axis, the bruit may be accentuated with expiration.

The lesion responsible for stenosis or occlusion of the arteries supplying the visceral circulation is an atheroma involving the ventral aspect of the suprarenal aorta. This lesion surrounds and overflows the orifice of the celiac and superior mesenteric arteries and extends a short distance into these aortic branches. More extensive circumferential aortic involvement may also be seen with contiguous involvement of the adjacent renal arteries and the infrarenal aorta, and the orifice of the inferior mesenteric artery. A collateral pathway occurs in response to progressive obstruction of one or more visceral branches (Fig. 1). The arterial connection between the visceral branches develops immediately distal to the orifice lesion, and blood flow is maintained to the distal vascular bed. Major pathways include: the gastroduodenal, connecting the celiac and superior mesenteric arterial systems in the duodenal-sweep and re-

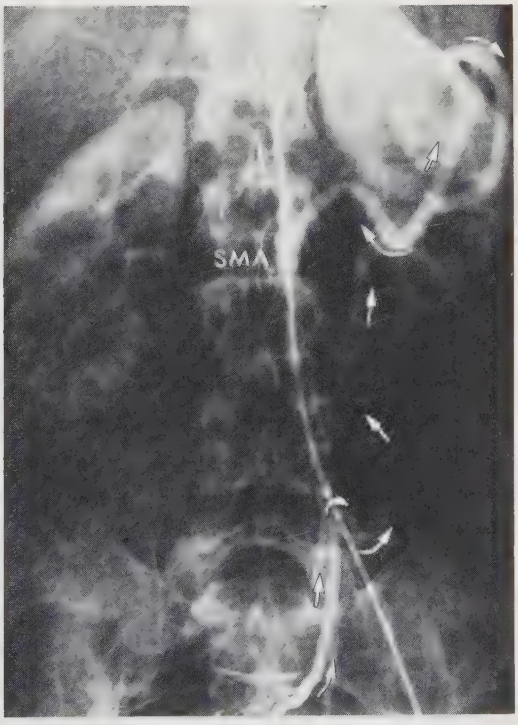


Figure 1. A large collateral vessel (arrows) originates from the pelvis (left hypogastric artery) and supplies the superior mesenteric artery through sigmoidal and mesenteric collateral vessels.

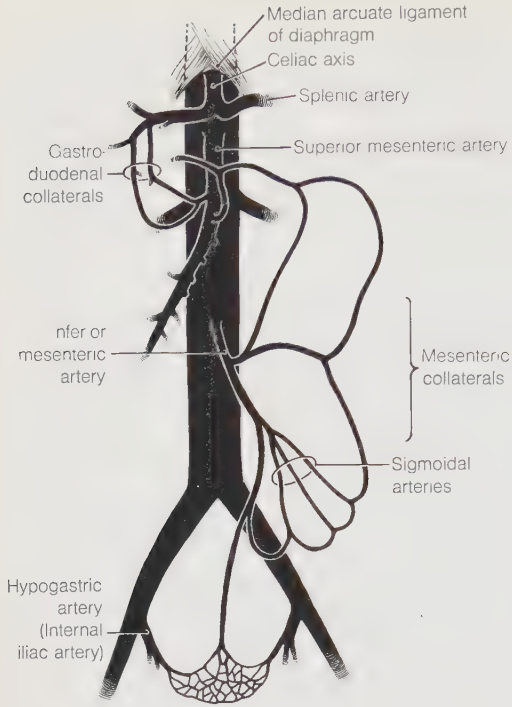


Figure 2. The major aortic branches supplying the gastrointestinal tract are shown. The collateral pathways are evident.

tropancreatic regions; the left colic or meandering mesenteric, connecting the inferior mesenteric and superior mesenteric arterial systems through the mesentery of the descending colon; and the sigmoidal, connecting the inferior mesenteric and internal iliac (hypogastric) arterial systems in the pelvis and the mesentery of the sigmoid colon (Fig. 2). Previous or contemplated gastrointestinal resection which traverses these anatomic regions may divide these protective vascular connections and in patients with occlusive lesions of the visceral branches may precipitate or aggravate vascular impairment.

Certain patients may remain asymptomatic despite major occlusive lesions because of the abundant mesenteric circulation supplied by these collaterals.[3] In the absence of an arteriogram in such a patient who requires surgical management of another gastrointestinal or aortic lesion, it is important to preserve the collateral pathways to avoid precipitation of acute intestinal ischemia. Intraoperative assessment of the mesenteric vessels and their collaterals should be performed to avoid compromising the visceral circulation. Consideration might be given to visceral revascularization in selected patients in whom resection of a lesion in the gastrointestinal tract would traverse established collaterals which are maintaining a viable intestinal circulation.

In patients with compression of the celiac axis, the lateral view shows an indentation of the anterior surface of the proximal celiac vessel, frequently associated with a poststenotic dilatation. However, athero-

sclerotic involvement of the celiac or other visceral vessels is usually lacking. Films exposed in full expiration will frequently show increased compression of the celiac artery, which will further narrow or occlude the lumen.

MANAGEMENT

Acute Ischemia

One of the initial steps in the management of acute intestinal ischemia is to obtain an arteriogram to allow the surgeon to make a definitive diagnosis. Simultaneously, measures should be instituted to monitor the patient properly, correct any hemodynamic instability, and treat any existing or anticipated infection. Some authors have recommended keeping the angiographic catheter in place and infusing a vasodilator while the patient is being prepared for surgery.[2]

The surgical management of either embolic or thrombotic occlusion has two phases: Revascularization of the intestinal tract and appropriate resection of the bowel, if indicated.[1] Embolic occlusion responsible for acute symptoms almost invariably involves the superior mesenteric artery. At laparotomy, the superior mesenteric artery is exposed at the base of the mesentery and opened through a transverse arteriotomy. The embolus is then removed with an appropriately sized balloon catheter. Small pieces of clot lying in distal branches may be flushed out with heparinized saline solution. The arteriotomy is then closed with interrupted 6-0 or 7-0 monofilament suture and mesenteric flow is restored.

Attention is then directed to the intestinal tract. Any obviously necrotic bowel should be resected. With the restoration of blood flow to the mesenteric vessels, marginal bowel may regain viability. A 30 to 45 minute period of observation of the bowel at this point will frequently demonstrate improved viability of much of the bowel, and thus extensive bowel resection can be avoided. We do not hesitate to use a "second-look" procedure. If there is some question about large segments of bowel, then only the frankly necrotic bowel is resected and a planned repeat laparotomy is performed at 24 hours. At this time the status of the remaining bowel will have declared itself and a final decision can be made regarding the necessity for further resection.

The superior mesenteric artery is also the vessel most frequently involved by acute thrombosis; the celiac and inferior mesenteric arteries may also be involved. As in patients with embolic occlusion, the first step is to restore the flow of blood. If the superior mesenteric artery is involved, then it is exposed along with the suprarenal aorta. The artery is opened through a longitudinal arteriotomy extended distally to the termination of the atherosclerotic lesion and proximally onto the aortic wall. An endarterectomy is then performed, with care taken to remove that portion of the lesion which involves the orifice of the superior mesenteric artery. After a satisfactory distal endpoint also has been obtained, the arteriotomy is then closed with a vein patch. An occluded or significantly

stenosed celiac axis may be revascularized by either endarterectomy or autogenous graft which originates from the anterior surface of the distal thoracic aorta and is anastomosed to the end of the celiac trunk. In our experience, grafts from the infrarenal aorta to the superior mesenteric artery, which have a retrograde alignment, have been prone to early thrombosis.

Subsequent to revascularization, the same consideration as described above should be given to bowel resection. Again, we prefer to be conservative regarding bowel resection at the time of the initial laparotomy and to employ a "second-look" procedure, if indicated.

The treatment of nonocclusive visceral ischemia should be aimed initially at treatment of the inciting event which produced a decrease in visceral perfusion—congestive heart failure, shock, digitalis toxicity, and so on. Resection may be necessary at this time to remove frankly necrotic bowel. The intra-arterial infusion of a vasodilator has been efficacious in decreasing the vasospasm and promoting mesenteric blood flow. As more experience is gained with nonocclusive visceral ischemia, it has become apparent that stenotic lesions involving the proximal visceral vessels are frequently present, thus making these patients more susceptible to ischemia of the bowel at the time of vasoconstriction.[6, 10] Therefore, as soon as the patient has recovered from the initial ischemic episode, serious consideration should be given to correcting any stenotic lesion in order to prevent a recurrence of intestinal ischemia.

Chronic Ischemia

The celiac axis compression syndrome is a result of extrinsic compression on the celiac axis by the median arcuate ligament. Proper treatment of this lesion necessitates dividing the ligament and returning the compressed artery to its normal caliber. The latter usually can be accomplished by passing dilators in a retrograde fashion through a transverse arteriotomy in either the splenic or hepatic artery. If this does not restore the vessel to its normal caliber, the proximal celiac artery may be replaced by a 6 mm Dacron graft.

The most challenging problems in visceral revascularization are those found in patients with chronic ischemia in whom multiple vessels are involved by atherosclerosis. Over the years, a number of techniques have been developed to restore blood flow to the mesenteric vessels.[4, 9, 11, 12, 14] Most of these have involved saphenous vein or Dacron grafts from the aorta to the celiac or superior mesenteric arteries. Some centers have recommended placing these grafts in a retrograde fashion.[4, 9] In our experience, saphenous vein grafts, especially those positioned in a retrograde fashion, have not proved to be durable. Certainly the mobility of the superior mesenteric artery in relation to the aorta makes grafting between the two difficult.

On the vascular service at the University of California, two procedures have proved to be reliable and durable. In patients in whom only the celiac artery can or needs to be revascularized, a 6 mm Dacron graft from the anterior surface of the distal thoracic aorta to the celiac artery

performed by the transabdominal route can easily be placed. The distal thoracic aorta and celiac axis are exposed through the lesser sac. The crus of the diaphragm is divided to allow exposure of the aorta which is free of atherosclerosis. Cross clamping of the aorta proximally and distally is recommended during the fashioning of the proximal end-to-side anastomosis. A distal anastomosis is performed end-to-end to the celiac artery beyond the diseased portion. Endarterectomy is recommended when reconstruction of both the celiac and the superior mesenteric arteries is required. Early in our experience, we preferred transabdominal endarterectomy; however, over the last five years we have utilized a retroperitoneal thoracoabdominal approach. This provides excellent exposure of the suprarenal aorta and allows for a complete endarterectomy of both the celiac and superior mesenteric arteries. Concomitant reconstruction of renal arteries or aortofemoral reconstruction may also be performed with this exposure.[12]

The thoracic portion of the exposure is developed through an incision in the eighth intercostal space that is extended obliquely across the abdomen beyond the midline. Retroperitoneal dissection permits retraction of the peritoneal contents to the right of the patient. The left crus and posterolateral and anterior fibers of the left hemidiaphragm are transected circumferentially adjacent to the chest wall to avoid denervation of the diaphragm, thus reducing the incidence of diaphragmatic dysfunction in the postoperative period. With this method the lower thoracic, entire abdominal and common iliac arteries are easily exposed (Fig. 3).

When only the celiac and superior mesenteric arteries are involved

Figure 3. A left transthoracic retroperitoneal exposure shows the entire abdominal aorta and its major branches.

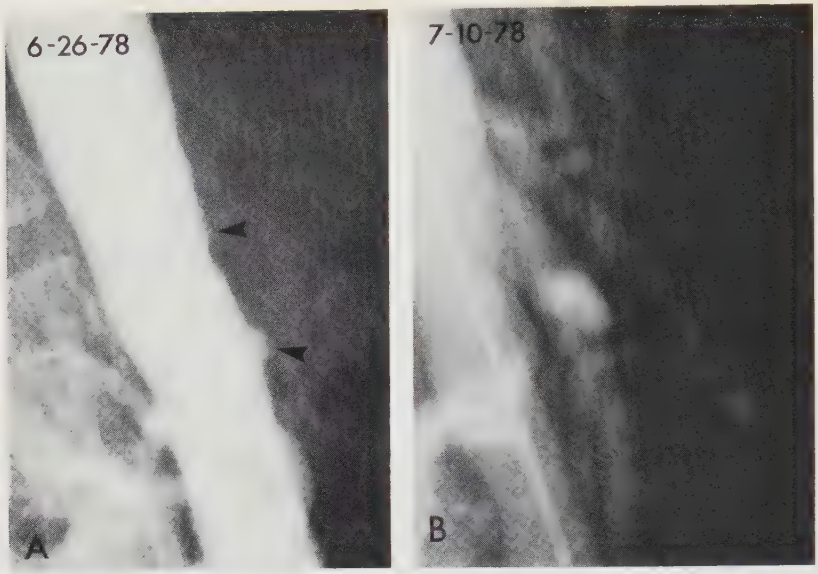


Figure 4. A 53 year old woman had abdominal angina. *A*, Arrows indicate the stumps of the occluded celiac and superior mesenteric arteries on the preoperative arteriogram. *B*, The celiac artery and superior mesenteric artery are widely patent following transaortic celiac and superior mesenteric artery endarterectomy.

by the atherosclerotic process, the aorta is opened with a longitudinal aortotomy placed to the left of these vessels. A transverse, hockey-stick incision at either end of the aortotomy creates a trap door that can be reflected medially to expose the orifices of the two mesenteric vessels. The endarterectomy is begun at the undersurface of the trap door and extended into the visceral vessels. Simultaneous traction on the specimen and instrumental separation of the intimal lesion causes the endarterectomized artery to prolapse into the aortic lumen, thus permitting the surgeon to visualize the end point. The aortotomy is then closed with a running suture. The duration of total aortic occlusion rarely exceeds 25 minutes (Fig. 4,*A* and *B*).

When the lesion has progressed to total occlusion of the superior mesenteric artery, the orifice portion of the lesion can be removed as described above and flow may be restored in the aorta with the proximal superior mesenteric artery still occluded. The distal artery opposite the first branches is opened longitudinally and the remainder of the specimen and adherent propagated thrombosis is removed under direct vision. The arteriotomy is then closed using a vein patch.

If the aortic lesion involves the renal arteries, the aortotomy may be extended longitudinally down the proximal infrarenal aorta to provide exposure for transaortic removal of these lesions as well.

No neurologic complications to suggest spinal cord ischemia have resulted from the use of this technique. One patient operated upon for severe visceral as well as renal insufficiency has been relieved of visceral ischemia, but chronic renal impairment persists.

RESULTS

The results of treatment of patients with acute mesenteric infarction, especially those with acute thrombosis, have been uniformly poor.[1, 2, 7] This is a result of a multiplicity of factors: delay in arrival of patients, delay in making the diagnosis, associated sepsis, and associated systemic disease, especially cardiac disease.

However, improved results are possible, particularly in patients sustaining an embolus in the mesenteric artery. Bergan's study has demonstrated that embolectomy prior to resection of the bowel is the treatment of choice.[1] Of 22 patients, only 2 of 11 were long-term survivors of primary resectional therapy alone, while 8 of 11 were survivors of embolectomy and resection if indicated. Also, in those patients undergoing resection without revascularization, lethal intestinal fistulas occurred in four.

Results of the treatment of celiac axis compression syndrome have been varied. Indeed, much controversy remains among surgeons regarding the efficacy of any procedure for this syndrome.[5, 8, 13, 14] A review of the arguments both pro and con is beyond the scope of this paper. Suffice it to say that we have been impressed that certain selected patients can benefit from celiac decompression.[8, 14]

We have recently reviewed our experience with 53 patients with chronic visceral ischemia secondary to atherosclerotic lesions. Techniques of revascularization included reimplantation, replacement grafting and endarterectomy.[11]

Reimplantation was utilized early in our experience with excellent results in two of the four patients so treated, but has generally been abandoned because it has limited applicability because of atheromatous involvement of the adjacent aorta that makes reimplantation impractical.

Replacement grafts were used in 14 patients. With one exception, these were antegrade grafts arising from the distal thoracic aorta. Three of the grafts were arterial autografts. Two of the three patients have been asymptomatic for six and seven years; one graft failed secondary to a technical error. The donor vessel in each was the external iliac artery; the hypogastric arteries were not used because of their contribution to visceral blood flow.

Five of six saphenous vein grafts failed within two years, yet all functioned satisfactorily earlier. This technique has now been abandoned in our institution and the saphenous vein is used only for patching a longitudinal arteriotomy in the superior mesenteric artery.

Dacron antegrade grafts (6 to 6.5 mm) were used in six patients with excellent results in all. Five of these were for the celiac artery and one for the superior mesenteric artery.

Endarterectomy was performed by either the transarterial or transaortic method. The latter utilized either a transabdominal or thoracoabdominal route. The transarterial technique was successful in the four patients so treated. However, this technique does not permit adequate removal of the aortic portion of the atheromatous lesion and has thus been supplanted by transaortic techniques. The transabdominal trans-

aortic approach was used in seven patients; six have had excellent results. However, the overlying pancreas restricts exposure, and the ventral orientation of the celiac and superior mesenteric arteries makes transaortic endarterectomy awkward from this approach. The technical drawbacks of previous methods are eliminated by the thoracoabdominal approach, which has been used in 12 patients, five of whom had simultaneous renal artery reconstructions. Eleven of the 12 patients have remained asymptomatic over a follow-up period ranging from one to seven years. One patient died from hepatitis six weeks postoperatively.

A review of our experience reveals that generally poor results may be anticipated when saphenous vein grafts are used to substitute for visceral arteries, whereas very satisfactory results were obtained using Dacron grafts. In patients with chronic intestinal ischemia, revascularization of the celiac axis was very important. Four patients had lesions involving both the celiac and the superior mesenteric artery. Both were repaired but the celiac reconstruction subsequently failed. One of these patients died from intestinal gangrene and two patients required a second operation on the celiac artery to relieve recurrent symptoms. Only one patient remained asymptomatic. Ten other patients had a similar pattern of involvement of the visceral arteries and had only one of the two vessels repaired. Of the seven patients in whom only the celiac artery was revascularized, six were subsequently asymptomatic. The one symptomatic patient was found to have a residual stenosis of the celiac artery at follow-up arteriography. Of the three patients in whom the procedure was confined to the superior mesenteric artery, two had persistent symptoms and subsequently required a second procedure to correct the celiac lesion. Five patients had involvement of three mesenteric arteries. Revascularization of the celiac and inferior mesenteric arteries was performed in four with relief in all. The superior mesenteric artery and inferior mesenteric artery were repaired in the fifth; he, too, was relieved of symptoms. Thus, in 19 patients in whom the celiac artery was involved, only three were free of symptoms when a residual lesion remained in the celiac artery. Conversely, a residual lesion in the superior mesenteric artery would appear to be benign providing the celiac and inferior mesenteric arteries are patent.

It would appear from reviewing these data that correcting the celiac lesion alone would be sufficient, providing the inferior mesenteric artery is spared. However, because patients frequently present at a young age and with a relatively extensive and rapidly progressive pattern of atherosclerosis, it remains our belief that both the celiac and superior mesenteric arteries should be repaired if it is technically feasible and if the patient is able to tolerate an extensive revascularization. Unrestricted access to the entire abdominal aorta and its branches is one of the greatest advantages of the retroperitoneal thoracoabdominal route.

SUMMARY

Acute and chronic visceral ischemia are now well recognized clinical entities and extensive progress has been made in both the evaluation and

treatment of patients with these lesions. The visceral vessels and the nature of their collateral pathways can also be of critical importance when dealing with other intra-abdominal pathology. Poor anastomotic healing, or the formation of strictures and intestinal fistulas may result when the mesenteric circulation is slowly compromised. Hence, it is imperative that the visceral arteries, their lesions, and their collateral pathways be understood by all surgeons operating in the abdominal cavity.

REFERENCES

1. Bergan, J. J., Dean, R. H., Conn, J. Jr., et al.: Revascularization in treatment of mesenteric infarction. Ann. Surg., *182*:430, 1975.
2. Boley, S. J., Sprayregan, S., Siegelman, S. S., et al.: Initial results from an aggressive roentgenological and surgical approach to acute mesenteric ischemia. Surgery, *82*:848, 1977.
3. Cheine, J.: Complete obliteration of the celiac and mesenteric arteries: Viscera receiving their blood supply through extraperitoneal system of vessels. J. Anatomic Physiol., *3*:65, 1868.
4. Crawford, E. S., Morris, G. C., Myhre, H. O., et al.: Celiac axis, superior mesenteric artery, and inferior mesenteric artery occlusion: Surgical considerations. Surgery, *82*:856, 1977.
5. Evans, W. E.: Long-term evaluation of the celiac band syndrome. Surgery, *76*:867, 1974.
6. Heer, F. W., Silen, W., and French, S. W.: Intestinal gangrene without apparent vascular occlusion. Am. J. Surg., *110*:231, 1965.
7. Krausz, M. M., and Manny, J.: Acute superior mesenteric arterial occlusion: A plea for early diagnosis. Surgery, *83*:482, 1978.
8. Lord, R. S. A., Stoney, R. J., and Wylie, E. J.: Coeliac-axis compression. Lancet, *2*:795, 1968.
9. Reul, G. J., Jr., Wukash, D. C., Sandiford, F. M., Surgical treatment of abdominal angina: Review of 25 patients. Surgery, *75*:682, 1974.
10. Russ, J. E., Haid, S. P., Yao, J. S. T., et al.: Surgical treatment of nonocclusive mesenteric infarction. Am. J. Surg., *134*:638, 1977.
11. Stoney, R. J., and Wylie, E. J.: Recognition and surgical management of visceral ischemic syndromes. Ann. Surg., *164* 714, 1966.
12. Stoney, R. J., Ehrenfeld, W. K., and Wylie, E. J.: Revascularization methods in chronic visceral ischemia caused by atherosclerosis. Ann. Surg., *186*:468, 1977.
13. Szilagyi, D. E., Rian, R. L., Elliott, J. P., et al.: The celiac artery compression syndrome: Does it exist? Surgery, *72*:849, 1972.
14. Wylie, E. J., and Stoney, R. J.: Management of visceral arterial occlusions. *In* Dale, W. A. (ed.): Chicago, Year Book Medical Publishers, 1971.

Department of Surgery
University of California
San Francisco, California 94143

Extraanatomic Bypass Grafts

James W. Holcroft, M.D.,[*]
Sebastian Conti, M.D.,[†] *and F. William Blaisdell, M.D.*[‡]

Although there are a number of different types of extraanatomic bypass graft procedures including those which can be used to revascularize cerebrovascular occlusive lesions, this paper will be limited to discussion of those procedures used to maintain or restore circulation to the lower extremities. These include axillofemoral bypass, femorofemoral bypass, and obturator-femoral bypass.

BACKGROUND

Freeman and Leeds should be credited with performing the first extraanatomic bypass graft procedure.[4] Their report, published in 1952, which presented a case of common femoral-to-common femoral bypass utilizing a segment of endarterectomized superficial femoral artery, was the first true extraanatomic bypass procedure reported in the surgical literature. Subsequently, McCaughan and Kahn,[7] Blaisdell et al.,[1] and Vetto[9] all independently reported on this technique. But the large successful series of Vetto should be credited with giving impetus to its adoption for general use.[9]

In 1960 we were confronted with what appeared at that time to be an insurmountable problem: infection in an abdominal Dacron bifurcation graft used to replace the abdominal aorta after resection of an aneurysm. Hemorrhage occurred from the proximal suture line of the graft approximately 30 days after the initial surgery. The original graft was then removed and replaced with another. Cultures taken of what appeared to be an infected field confirmed the presence of bacteria. Despite intensive treatment, the patient remained septic, and the second graft ruptured at the proximal anastomosis 30 days following its

[*]Assistant Professor of Surgery, Department of General Surgery, University of California-Davis School of Medicine, Davis, California
[†]Assistant Professor of Surgery, Department of General Surgery, University of California-Davis School of Medicine, Davis, California
[‡]Professor of Surgery and Chairman, Department of General Surgery, University of California-Davis School of Medicine, Davis, California

placement. At this point, the abdomen was opened, the proximal aorta and iliac arteries were ligated, and the bifurcation graft was removed. A Dacron tube was anastomosed to the thoracic aorta through a small thoracotomy incision and was brought down the lateral side of the abdomen to the groin, then anastomosed to the left femoral artery. Placement of the femoro-femoral graft completed the revascularization of both legs. This case confirmed the validity of the concept that a small Dacron tube could support the circulation to the lower half of the body and that extraanatomic reconstruction could be successfully substituted for the abdominal aorta.[1] However, the use of the thoracic aorta for proximal insertion of the bypass graft was not optimal since it involved carrying out thoracotomy in a critically ill patient. Hence, the next time we had occasion to carry out an extraanatomic bypass procedure, we elected to use the proximal axillary artery as it emerged from the clavicle as the inflow vessel; we tunneled the graft down the side of the abdomen subcutaneously and then anastomosed this to the femoral artery.[2] The procedure was successful for 2½ years until the patient's demise from a myocardial infarction. Simultaneous with the development of our procedure, Louw[6] carried out a similar procedure and also reported successful results.

Another major advance in the use of extraanatomic grafts was reported by Shaw and Baue in 1963.[8] Their procedure involved rerouting the bypass graft through the obturator foramen in order to bypass an infection in the graft at the groin. This anatomical route proved to be an excellent one for bypassing the inguinal region when infection was limited to this area.

Since our initial experience in 1960 with extraanatomic bypass, we have accumulated 19 years of experience with these procedures and believe their use can now be put into proper perspective.[3]

INDICATIONS FOR OPERATION AND SELECTION OF THE SPECIFIC BYPASS PROCEDURE

Extraanatomic arterial bypass grafting is used when the risk of standard operations is high because of associated disease or anticipated technical problems. The most common indication for this type of operation is severe pulmonary or cardiac disease. A patient with myocardial infarction, low cardiac output and hypotension may develop gangrene or severe ischemia of extremities as a result of preexisting aortoiliac occlusive disease or embolism from a mural cardiac thrombus. In this circumstance, femoro-femoral bypass or axillo-femoral arterial bypass can be performed under local anesthesia or light general anesthesia with less risk than is associated with amputation.

The specific vascular problems for which these extraanatomic arterial bypass operations have been of value are aortoiliac occlusive disease, aneurysms of the aortoiliac system, and infection of the peritoneum, retroperitoneum or inguinal regions. Aortoiliac occlusive disease should be treated by conventional procedures when the risk to the pa-

tient is reasonable. But if preexisting pulmonary or cardiac disease is thought to produce a risk of mortality greater than 5 per cent, an extraanatomic bypass procedure may be indicated. Abdominal vascular disease may be satisfactorily bypassed by using the contralateral femoral or the ipsilateral axillary artery as the source of inflow, the choice between these two depending upon whether the disease is unilateral or bilateral. The contralateral axillary artery can be used if the disease exists in the ipsilateral subclavian artery. In poor-risk patients, these bypass operations avoid systemic morbidity, since they can be carried out on the surface of the body. Technical problems when they occur are local, and even prolonged operations are well tolerated.

Although we have treated several patients having abdominal aneurysms using axillo-femoral bypass, these procedures must be considered experimental. An axillo-femoral graft that is placed in parallel with the aneurysmal aorta will decrease flow through the aorta. In theory, this should increase the laminated clot in the abdominal aorta and narrow the aortic lumen. When this procedure has been performed for pain, symptoms have disappeared in these patients. But for obvious reasons, when the patient is a candidate for conventional aneurysm resection, the standard operation should be carried out.

The most pressing indication for an extraanatomic bypass procedure—and this is an absolute one—is infection in a previously placed aorto-iliac graft. In this instance, the treatment of choice is total removal of the infected graft with ligation of the infrarenal aorta and iliac arteries. Bypass of the abdominal infection is then performed with axillo-femoral or femoro-femoral grafts, or a combination of the two. When vascular occlusion occurs together with an intraabdominal catastrophe such as peritonitis resulting from diverticulitis, femoro-femoral or axillo-femoral arterial grafts can be used to bypass the contaminated or infected abdomen. If infection is limited to one groin such as in the instance where a common femoral aneurysm has been replaced with a graft, obturator bypass is the procedure of choice and provides a means of circumventing the infected area through a very satisfactory alternate anatomic route.

CONTRAINDICATIONS TO BYPASS PROCEDURES

There are very few contraindications to the use of extraanatomic bypass grafting procedures. They are essentially the same contraindications as for any vascular reconstructive procedure: inadequate inflow or outflow vessels, or infection along the proposed route of the graft.

Blood pressure should be carefully measured in both arms, and the subclavian area should be auscultated, as decreased blood pressure in one arm may contraindicate use of the corresponding arm. A bruit or decreased brachial artery pressure should be evaluated with arteriography to rule out disease of the proximal subclavian artery. A stenosis of the innominate or subclavian artery of 50 per cent or more rules out use of the proximal axillary artery as the source of inflow.

A patent profunda femoral artery is the minimum requirement to provide outflow to an axillo-femoral or femoro-femoral graft, while a patent superficial femoral or popliteal artery is required for adequate outflow of an obturator bypass graft. If preoperative arteriography has not demonstrated the outflow tract, operative arteriography must be performed to ensure that this requirement will be met and that runoff will be adequate.

OPERATIVE TECHNIQUE

Although these procedures have been carried out successfully under local anesthesia, a light general anesthetic is usually the anesthesia of choice. Graft materials may consist of autogenous vein, woven or knitted Dacron, or the newer polytetrafluoroethylene grafts. Generally, we have favored a knitted Dacron graft of the external velour type because of its size, ready availability and proven durability. Knitted Dacron tubes 10 mm in diameter are considered optimal for use as axillo-femoral grafts, although 8 mm Dacron tubes can be used successfully as well. Eight or 10 mm Dacron tubes are optimal for femoro-femoral grafts or for aorto- or ilio-transobturator superficial femoral bypass grafting. If there are two operating teams, bypass procedure can be performed in less than one hour.

Femoro-Femoral Bypass

Femoro-femoral bypass is the procedure of choice when dealing with occlusive disease involving one iliac system or when dealing with an aorto-iliac or aorto-femoral graft in which one limb is patent. The common femoral artery is the most accessible donor vessel, but the external iliac or deep femoral artery can also be used. A Dacron tube can be carried preperitoneally by tunneling under the inguinal ligament or by tunneling subcutaneously across the pubis to the opposite vessel. The anastomoses are made with an obliquely cut end of the Dacron graft to avoid narrowing at the anastomosis. Orientation of the graft in a transverse direction or at a reverse oblique angle from the femoral artery has not limited flow, nor has it seemed to increase the graft thrombosis rate. Since the reconstruction parallels flexion creases, the graft rarely kinks or obstructs (Fig. 1).

Axillo-Femoral Bypass

Axillo-femoral bypass has been used by choice when there is occlusive disease or both iliac systems or when infection is present. The axillary artery is a satisfactory inflow vessel if no proximal occlusive disease of the corresponding subclavian artery is present. Verification of patency by arteriography is optimal but not essential. The axillary artery itself is rarely diseased and its proximal one-third between the clavicle and pectoralis minor muscle is the segment of choice for proximal anastomosis. The first portion of the artery is fixed to the chest wall so that the anastomosis does not flex with motion of the arm.

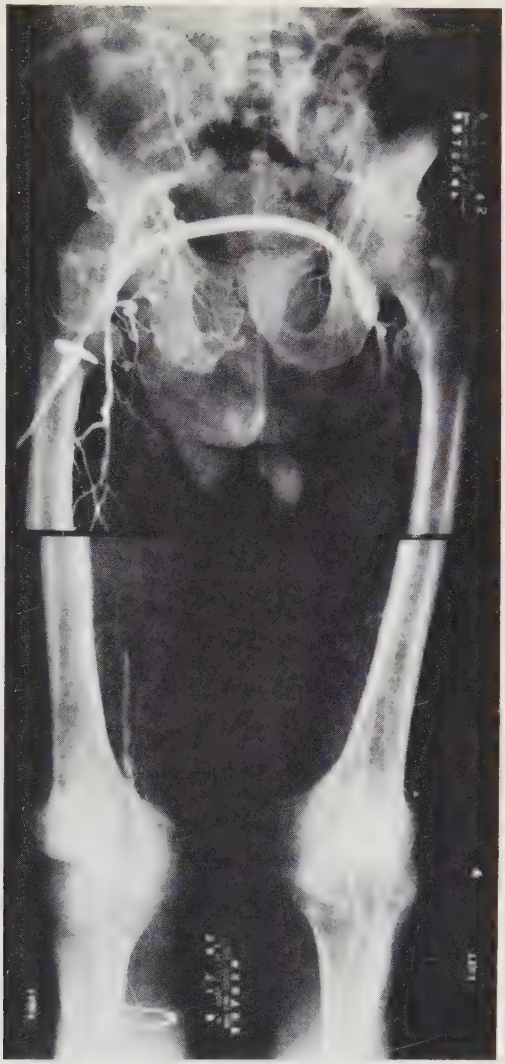


Figure 1. Femoro-femoral bypass. The retrograde curve of the proximal anastomosis has not seemed to compromise flow or affect immediate or late patency. The anastomoses are best placed in such a fashion as to bend gradually and lie comfortably in the groin.

There is only one small collateral branch from this segment, the highest thoracic artery. The tunnel for the graft is created behind the subclavian vein deep to the pectoral musculature downward and laterally to the midaxillary line, where a small counterincision at about the level of the eighth to tenth rib facilitates development of a tunnel to the groin. The lower portion of the tunnel can be carried subcutaneously or deep to the external oblique fascia. This tunnel is best carried posterior to the anterior axillary line at the waist so that angulation or kinking is less likely to occur when the patient sits or bends. End-to-side anastomosis to the common femoral or profunda femoral artery is then carried out in conventional fashion. If the ipsilateral ax-

Figure 2. Axillo-femoral bypass can be carried out using either axillary artery, although we favor the right artery because it has a lower incidence of proximal disease. At present bilateral rather than unilateral procedures are considered to be preferable because of better long-term patency.

illary artery is diseased, the contralateral artery can be used. In this instance, the graft is brought down to the level of the ipsilateral anterior iliac spine and curved transversely across the lower abdomen to the opposite femoral artery (Fig. 2).

The procedure which maximizes longevity of the axillo-femoral graft is a combination of axillo-femoral and femoro-femoral bypass grafting. This doubles the flow in the axillo-femoral section of the graft and improves long-term patency of the graft over that obtained with unilateral axillo-femoral procedure.[3, 5] In carrying out this procedure, one graft is first carried from the axillary artery to the corresponding femoral artery. If a bilateral procedure is to be done, a second graft is

then anastomosed to the first in the lower abdomen just below the inguinal ligament and as close to the lower anastomosis as possible, and is carried to the opposite femoral vessel subcutaneously or preperitoneally. This reconstruction provides total substitution for the infrarenal abdominal aorta and permits retrograde perfusion of the hypogastric artery if one or both external iliac arteries are patent.

Obturator Foramen Bypass

In the obturator foramen bypass the abdominal aorta, the ipsilateral or contralateral, common iliac, or external iliac arteries may be used as the proximal inflow vessel. A transperitoneal or retroperitoneal approach to the inflow vessels can be used. We use a small lower abdominal oblique incision when the proximal end of the graft is to be anastomosed to the iliac artery. The graft is tunneled through the upper medial portion of the obturator foramen. The obturator nerve and artery pass through this particular region, so it is essential that care be taken to protect the vessel and nerve as the fascia is divided along the inferior aspect of the pubic ramus. A hole is then made through the obturator membrane that is sufficiently large to admit the index finger, and a tunneler is passed down the plane of the adductor musculature to the level of the adductor canal. This puts the tunnel in immediate proximity to the distal portion of the superficial femoral artery just above the adductor magnus muscle, well away from any infected groin incision. Conventional end-to-side anastomosis can then be carried out at this level or the graft can be tunneled further down into the popliteal fossa for anastomosis with the popliteal artery. The advantage of this route is that it passes through the axis of flexion of the body so there is no tendency for the graft to kink when the patient assumes a sitting position. When infection is present, autogenous vein is the grafting material of choice.

Upon completion of all of these grafting procedures, we as a matter of policy carry out operative arteriography to document patency prior to closure of the incisions.

POSTOPERATIVE MANAGEMENT

The only special postoperative precaution for bypass grafting is prevention of flexion of the thighs within the immediate postoperative period. Vital signs should be monitored carefully; the pulse in the graft and the status of the feet are also monitored. Doppler pressures are recorded in foot vessels both preoperatively and postoperatively since a significant improvement in Doppler pressures is expected if the surgery is successful. No special medications are administered. If antibiotics are indicated for management of graft infections, they should be initiated 24 hours prior to operation, administered intravenously during the procedure, and then continued for 24 to 48 hours postoperatively.

All of our patients with bypass grafts are placed on antiplatelet-

aggregating drugs since this therapy has the theoretical advantages of preventing arterial clot and slowing the progression of arteriosclerosis. Enteric-coated aspirin is administered orally starting on the second to third postoperative day and is continued indefinitely at a dosage of 600 mg daily.

COMPLICATIONS

The complications of extraanatomic bypass procedures are similar to those associated with any other major vascular operation. These consist primarily of thrombosis and infection. If thrombosis should occur, reoperation is easily done under local anesthesia. However, restoration of patency must be verified by operative angiography involving both anastomoses and the entire graft. If infection occurs, this is generally an indication to remove the graft, ligate the inflow and outflow vessels, and accept amputation if this is necessary. This is preferable to risking the patient's life as a result of hemorrhage from a suture line.

A theoretical consideration is whether or not small arteries such as the axillary or femoral arteries can supply adequate amounts of blood to the lower half of the body without some sort of "steal" phenomenon. Our experience with several hundred operations in which the axillary artery was used to supply blood flow to the lower half of the body has repeatedly verified the safety of the procedure. No cases of steal have been documented, ischemia of the upper extremities has not occurred, and when the operation has been technically successful, the circulation in the lower extremities has subsequently improved.

At first we feared an axillo-femoral graft might be constricted by tight clothing or by direct pressure of the body on the graft while the patient was sleeping. But these fears were apparently unwarranted since despite warnings to the contrary, patients have continued to wear belts and have obviously slept lying directly on the grafts without causing constriction. We have not seen any graft thromboses that could be directly related to this sort of compression. In fact, intentional attempts to occlude the graft by digital pressure during arteriography are usually unsuccessful. This leads us to believe that when the blood pressure is normal it is very difficult to occlude one of these long grafts sufficiently to produce thrombosis.

RESULTS

Acute graft failures do not occur if arteriography demonstrates good flow in the graft and if anastomoses are free from technical defects. Therefore, we believe that all graft failures which occur in the immediate postoperative period are due to technical errors and are hence preventable.

Late failure cannot be accurately predicted since an occasional patient with very poor outflow has ultimately done well. However, patency seems to correlate with flow. That is, when flow is high through the superficially placed graft, the graft remains soft, thin and compressible. In this circumstance, long-term patency has been the rule. But when the flow rate through the graft is low, the graft becomes firm to palpation as a result of thickening of the neointima. We postulate that graft failure is due to disruption of the neointima as described by Wesolowski,[10] or to actual embolism occurring in portions of thickened intima as it becomes disrupted at the sites of flexion. A few of our grafts have remained patent for prolonged periods even though flow through them has been negligible. This supports the hypothesis that accidents to the neointima tend to produce most of the closures. The thicker the neointima, the greater seems the likelihood of these accidents.

As regards femoro-femoral grafts, long-term patency has been excellent and comparable to that for aorto-iliac bypass: a two-year patency rate of 80 per cent. Even when one or more outflow vessels are occluded, the femoro-femoral grafts have done comparably as well as the aorto-femoral grafts.

The patency rate is lower for axillo-femoral grafts and is similar to our experience with femoro-popliteal Dacron bypass grafts: a failure rate of 50 per cent at two years. The advent of the newer velour prostheses offers promise for more rapid healing and for improved patency. But our experience with these newer prostheses, while favorable to date, has not been sufficient to document whether or not they constitute a significant advance as regards long-term patency.

The risk of bypass operations is low and mortality has related to systemic disease rather than to the operative procedure itself.

We have not yet used axillo-femoral grafts in a series comparable to that of our aorto-femoral grafts. All of our patients had either serious systemic disease or life-threatening graft infection.

In LoGerfo's experience with 88 aorto-bilateral iliac or femoral grafts and 56 axillo-bilateral femoral grafts performed electively for the management of occlusive disease of the abdominal aorta or iliac vessels, the long-term patency has been excellent.[5] His group found that their results indicated the axillo-bilateral femoral graft, when used in an older population, had a lower operative mortality than did conventional aortic bypass procedures and had a similar long-term patency rate (76 per cent) at five years. They found, however, that axillo-femoral grafting required more frequent reoperation to obtain this patency rate.

We have not had sufficient experience with aorto-transobturator femoral bypass to present any statistical results. However, our experience to date with approximately 10 cases suggests that patency rates are equal to those for conventional bypasses when conventional grafting materials are used and the procedure is carried out along a parallel route. No large series have been reported which contradict our experience or results.

REFERENCES

1. Blaisdell, F. W., DeMattei, G. A., and Gauder, P. J.: Extraperitoneal thoracic aorta to femoral bypass graft as replacement for an infected aortic bifurcation prosthesis. Am. J. Surg., *102*:583–585, 1961.
2. Blaisdell, F. W., and Hall, A. D.: Axillary-femoral artery bypass for lower extremity ischemia. Surgery, *54*:563–568, 1963.
3. Blaisdell, F. W., Hall, A. D., Lim, R. C., Jr., et al.: Aorto-iliac arterial substitution utilizing subcutaneous grafts. Ann. Surg., *172*:775–780, 1970.
4. Freeman, N. E., and Leeds, F. H.: Operations on large arteries. Calif. Med., 77:229, 1952.
5. LoGerfo, F. W., Mannick, J. A.; et al.: A comparison of the late patency rates of axillobilateral femoral and axillounilateral femoral grafts. Surgery, *81*:33–40, 1977.
6. Louw, J. H.: The treatment of combined aorto-iliac and femoro-popliteal occlusive disease by spleno-femoral and axillo-femoral bypass grafts. Surgery, *55*:387, 1964.
7. McCaughan, J. J., Jr., and Kahn, S. F.: Cross over graft for unilateral occlusive disease of the iliofemoral arteries. Ann. Surg., *151*:26, 1960.
8. Shaw, R. S., and Baue, A. E.: Management of sepsis complicating arterial reconstructive surgery. Surgery, *53*:75–86, 1963.
9. Vetto, R. M.: The treatment of unilateral iliac artery obstruction utilizing femorofemoral graft. Surgery, *52*:342, 1962.
10. Wesolowski, S. A.: The healing of vascular prostheses. Surgery, *57*:319, 1965.

Room 257
UCD Professional Building
4301 X Street
Sacramento, California 95817

Management of Complications After Arterial Reconstruction

*D. Emerick Szilagyi, M.D.**

The variety of complications that can occur after reconstructive operations for arterial disease is vast. To assure a discussion in reasonable depth within the confines of a brief article the scope of review must be limited. In what follows only those complications will be touched upon which are directly traceable to a vascular intervention and are peculiar to it. Systemic and nonvascular organ-related complications after vascular surgical procedures, at any event, require no consideration other than that given to similar complications that follow nonvascular operations of like magnitude. Second, space limitations will restrict the exposition to statements of principle at the expense of detailed technical descriptions.

It will serve the clarity of presentation to group these complications under the headings of "early" and "late." Arbitrarily one defines early complications as those that occur before the completion of clinical healing and which are related to events occurring in the intraoperative or immediate postoperative period. Late complications result from events that occur after the immediate postoperative period and become manifest after clinical healing has run its course.

EARLY COMPLICATIONS

Early complications requiring secondary repair after reconstructive arterial operations are not seen often. In a group of 4247 patients treated between 1951 and 1973 the incidence of these complications was 2.9 per cent, or less than one-third of the frequency of late complications.[3]

These complications may represent events that have arisen within the anatomic limits of the surgical procedure ("in situ" complications), or structural damage and functional disturbance resulting from the ef-

*Department of Surgery, Henry Ford Hospital, Detroit, Michigan

fects of the surgical procedure but located at some distance from it ("ex situ" complications).

In Situ Complications

In situ complications comprise hemorrhage, thrombosis, and infection.

Hemorrhage. Early postoperative hemorrhage, a rare complication in well-executed present-day vascular surgery, is usually the consequence of some technical oversight. Abnormalities of the coagulation mechanism, present preoperatively or induced intraoperatively, may also be responsible. In both instances, prevention is the most effective means of treatment. Identification and correction of hematologic aberrations preoperatively and proper management of blood loss during the operation are important preventive means. The maintenance of normal blood pressure at the completion of the vascular procedure and during closure will help avoid overlooking an insecure anastomotic suture line or improperly controlled bleeders.

Thrombosis. Thrombosis at the site of reconstruction which has been properly conducted by experienced hands is rarely the result of technical errors. The cause is much more likely to be poor case selection, that is, overextension of the use of the given procedure. The distinction is of great importance since a technical error can, and promptly should, be corrected while the failure of a procedure because of its inapplicability in a given situation is beyond repair. The experienced surgeon is able to assign the correct mechanism for failure in most cases from the consideration of the preoperative angiographic and intraoperative anatomic findings. His judgment may be helped by immediate postoperative angiography and blood flow measurements. When the presence of a correctable technical imperfection is established, reoperation must be carried out. The frequency of this type of intervention in our experience is about 1 per cent. Rarely, early exploration with or without previous angiographic studies may be advisable even though either no technical error is suspected or its nature is not clear.

Infection. The clinical manifestations of wound-healing complications of importance including infection after primary vascular procedures, usually occur in the late postoperative period, even though their inception, with rare exceptions, takes place intraoperatively.[6] An important but rare exception is the hematogenous localization of a postoperative systemic infection at the site of a reconstructive operation or in the wall of an arterial prosthesis. The subject of treatment of infection in the site of a reconstructive vascular procedure can therefore be best discussed under the heading of late complications.

Manifest early infections do take place in the wounds of reconstructive vascular operations, but unless the suture line of repair or the body of the synthetic implant is involved, the management is not different from that of any wound infection. In case the early infection has reached the level of the prosthesis, the treatment follows the line of action to be described for late infections, with the minor difference that in some instances of early infection a trial with intensive antibiotic medication is permissible, particularly when the precise extent of

the infection is not known but the suture does not appear to be involved.

Ex Situ Early Complications

Either the temporary interruption by clamping or the intraoperative or early postoperative thrombosis of a critical artery may lead to complications of great importance.

Carotid Arterial Operations. Since all of the reconstructive procedures performed on the internal and common carotid arteries require interruption of the hemispheric blood flow, they carry the risk of neurologic damage. Since once the complications of a neurologic loss has taken place little, if anything, can be done to influence its effects, the question of prevention has a great practical importance. A rational plan of safeguards can be based on the following considerations.

Tolerance for the reduction of blood flow to the cerebral hemisphere by interruption of the internal carotid artery varies from patient to patient. Clinical experience has shown that even severe deprivation of blood is harmless for a period not exceeding four minutes. The ability of a patient to tolerate longer periods of interruption can be measured with reliable accuracy by the determination of blood pressure in the previously clamped internal carotid artery. The measurement is usually carried out by clamping the exposed common and external carotid arteries and determining the interluminal pressure (so-called "stump pressure") in the former, proximal to its bifurcation but distal to the clamp, by means of an electromagnetic flow meter. A stump pressure of 50 mm of mercury or higher has been found to indicate a collateral circulation to the cerebral hemisphere sufficient to allow clamping of the common carotid artery for prolonged periods of time.

In accordance with the considerations listed, the vast majority of surgeons have adopted one of two possible plans of preventive practice: either they disregard pressure measurements and use a shunt or a stent in every instance to maintain blood flow to the brain during the operative procedure, or they select patients for the use of a temporary bypass in accordance with the results obtained by blood pressure measurements. The surgeons who do not use one or another of the preventive forms of approach make up a very small minority. We have found the selective use of the Javid shunt in cases in which the stump pressure is below 50 mm of mercury entirely satisfactory. The employment of this method has allowed us to maintain a low incidence of permanent (1 per cent) or transient (2 per cent) serious neurologic complications.

As previously hinted, once a postoperative neurologic loss develops, its course can only slightly, if at all, be influenced by therapeutic measures. General supportive management and early physical rehabilitation are the only available form of treatment.

Renal Arterial Operations. Early renal complications consist of damage to the renal parenchyma that takes place during the surgical procedure or immediately thereafter.The factors leading to such injury may be listed as follows: presence of preoperative nephropathy, prolonged clamping of the aorta distal but close to the renal arteries,

normothermic interruption of blood flow to the kidney in excess of 30 minutes, encroachment on the ostium of renal artery by sutures, intraoperative arterial hypotension, and excessive blood transfusions.

The most important therapeutic principle in the management of renal complications is their avoidance. Once the damage has been established, the central problem of treatment is the control of uremia of various degrees.

Ureteral Problems. Though not specific to vascular operations, certain types of damage to the ureter occur with some frequency during procedures on the abdominal aorta and iliac arteries. The damage is related to extensive mobilization of the ureter, or to the stripping of the connective sheath carrying the blood supply to the ureter, or to the entrapment of the ureter under the prosthetic implant. One of the benefits of routine postoperative aortography is the observation of the effects of such injury that may otherwise go unnoticed. Once the existence of obstructive changes in the ureter is ascertained, careful follow-up by intravenous pyelography is necessary. Progressive changes require appropriate correction.

Colorectal Ischemia. A most interesting although uncommon early postoperative complication is colorectal ischemia.[1] Some degree of visually detectable ischemic change in the rectosigmoid can be observed during about 10 per cent of all procedures performed for resection of abdominal aortic aneurysms but only a small fraction of these patients show symptoms postoperatively. The predisposing cause is the simultaneous, either temporary or permanent, interruption of the inferior mesenteric and both internal iliac arteries, which, of course, is an unavoidable necessity of the operative procedure for resection of an abdominal aortic aneurysm. When the interruption is of short duration only mild changes will take place, consisting, at most, of superficial ulceration of the mucosa. The ischemic changes may, however, be much more severe, even reaching the degree of complete necrosis of the rectosigmoid segment. The most important aspect of management is prevention by reducing the length of time of occlusion of the hypogastric arteries and by always making sure that at least one internal iliac artery remains patent at the conclusion of the vascular procedure. In the typical instance of colorectal ischemia following surgery for an aneurysm the ischemic changes are limited to segments of various lengths of the distal colon. When, however, a previous operation may have interrupted the marginal artery of Drummond at a level approximate to that of the inferior mesenteric artery, clamping or ligation of the iliac arteries may lead to extensive necrosis even involving the entire left colon. If colorectal ischemia is suspected, the patient's postoperative course must be carefully watched for the presence of bloody blowel movements, and periodic rectal examination and proctoscopic visualization must be carried out. If the presence of ischemic changes is established, reduction of the bacterial content of the stool with appropriate oral antibiotics appears to be useful. Rarely a diverting transverse colostomy and even more rarely resection of part of the colon are necessary.

Ischemia of the Spinal Cord. Paraplegia caused by ischemia of the spinal cord during operations on the abdominal aorta is an extremely rare but disastrous complication.[4] Its incidence is about one in 400 aortic operations. A careful analysis of the anatomical background and of the clinical characteristics of this unfortunate phenomenon proves beyond doubt that in essence it is unforeseeable and therefore unpreventable. The anatomic circumstance that determines its occurrence is an anomalous greater radicular artery, the principal source of blood supply to the lower thoracic and to the lumbar spinal cord. When this artery has an abnormally low origin, it may become occluded during the placement of the aortic clamp. Unfortunately, such an arterial anomaly can only be detected preoperatively by a special angiographic study in which the lumbar arteries are selectively catheterized, an entirely impractical procedure both because of its technical difficulties and because of its considerable risk. Nor is it possible to recognize such an anomalous artery during operative dissection — indeed the artery requires great care for its demonstration even in the anatomist's dissecting laboratory.

Supportive and rehabilitative measures are the only possible therapy. The cord lesion fortunately is at times incomplete. In these cases, particularly those in which the posterior cord segments are spared, there is a fair probability of partial recovery.

Ex Situ Thrombosis Incidental to Vascular Resection. Thrombosis of a critical artery remote from the operative site may occur during arterial reconstruction which will require immediate surgical intervention. The most serious such complication is thrombosis of the superior mesenteric artery, which is peculiar to operations on the upper abdominal aorta and is caused either by misapplication of clamps or improper and extensive retraction. Its postoperative recognition is very difficult and calls for immediate surgical correction.

Thombosis of peripheral arteries during aortic operations can be readily prevented by adequate systemic heparinization. Emboli of atheromatous material from the operative site are usually small and do not require extraction.

LATE COMPLICATIONS

Late complications after arterial reconstruction are relatively common. In a group of 4247 cases we have recently surveyed, 10.2 per cent required secondary operations for the treatment of late complications. The incidence of late complications was about three and one half times that of early complications.[3]

Complications of Graft Healing

Healing complications after reconstructive vascular operations may be brought about by changes in the grafts themselves, or changes in the tissue environment of the graft.

Changes in Grafts

With our selection of prosthetic material virtually limited to Dacron, and the use of Dacron confined almost entirely to the aortoiliac area, we see few late complicatons caused by changes in prosthetic implants. Aortoiliac Dacron grafts do develop morphologic changes during the early months of their implantation but these changes seldom lead to loss of patency; they cause less than 5 per cent of late failures in these operations. In case they do fail, the occlusion cannot be corrected by thrombectomy but a new in situ (preferably, if conditions permit) or ex situ (extraanatomical) graft must be placed. In the surgical repair of an occluded aortoiliac or aortofemoral prosthesis an important technical detail is that the closed implant must not be excised. Instead, only the segments of the old graft near the anastomotic sites are removed to make room for the new anastomoses, and the new graft is placed over the old one without disturbing the latter.

The graft changes that currently play an important role in the late success of reconstructive operations occur in autogenous vein implants. Our information regarding the postimplantation behavior of these grafts is largely limited to the femoropopliteal area where the greater saphenous vein is used almost exclusively in the treatment of occlusive disease. Observations on these grafts are probably applicable, however, to other anatomical regions as well.

In a group of 260 cases of femoropopliteal autogenous vein bypasses for atherosclerotic occlusive disease, we investigated the changes in the implants by angiographic and histologic studies and found that although about 30 per cent of the grafts showed changes detectable in angiograms, most of the abnormal findings were minor.[2] In histologic studies the most consistent changes were atherosclerosis and subintimal thickening, both of which appear to be caused by the altered physiologic conditions of the vein when it is placed in the arterial circuit. About one-third of the changes observed were preventable because they were the result of technical mishaps, another third were suitable for secondary correction, and only about one-third or 10 per cent of the total number of vein grafts were of intrinsic nature and essentially irreparable. The important lesson we learned from this study was that venous implants must be carefully followed both by clinical and by angiographic means. Whenever postimplantation changes are detected, they must be considered for secondary surgical repair.

Complications Resulting from Changes in Tissue Environment

It is convenient to include under this heading false aneurysm, infection, and the formation of paraprosthetic fistulas. It must be noted, however, that a sizable proportion of false aneurysms originate as the result of changes in the recipient arterial wall rather than of those in the tissue environment.

False Aneurysms. These lesions constitute the single most common group of late postoperative complications.[5] They occur in slightly more than 1 per cent of all vascular anastomoses postoperatively. More than 95 per cent of the lesions are found in the area of the com-

mon femoral artery following aortofemoral bypass procedures with Dacron prostheses. For the sake of brevity in the subsequent remarks the subject of discussion will be solely this lesion.

The causative factors are complex. The most common factor appears to be the deterioration over the years of a recipient artery. Consequently, as the postoperative observation period lengthens one sees false aneurysms with greater frequency. Other contributing factors are subclinical infection (that is, infection that does not manifest itself in clinical signs and symptoms but interferes sufficiently with the healing process to prevent the proper sealing of the suture line of the anastomosis), disruption of the fabric of the prosthesis, mechanical abuse (excessively forceful physical activity), and breaking of the suture line. Hypertension contributes to the process but usually first some other factor is at play.

The treatment of false aneurysms of vascular anastomoses is primarily surgical. The indications depend on the symptomatology and on size. In the femoral area, we have found that clinically silent false aneurysms less than 2½ cm in diameter can be observed with safety, provided the follow-up is careful and includes the use of echography every three to six months and angiography at least once a year. A definite, although small, number of these lesions prove to be stable and will not require surgical intervention. Such lesions are probably not false aneurysms but represent local dilatation of the recipient artery, which in angiograms at times is impossible to distinguish from a false aneurysm. If the aneurysm under observation shows any increase in size surgical intervention becomes obligatory. It must be emphasized that the treatment of asymptomatic false aneurysms that have been electively submitted to operation are excellent and recurrence is rare. On the other hand, ruptured false aneurysms carry a significant morbidity and mortality.

The technical details of repair of a common femoral false aneurysm are determined by the anatomical features of the lesion. Occasionally it is possible to repair a false aneurysm by merely excising a small sac that has formed through an area of extravasation and stitch the defect in the suture line. Somewhat more commonly the false aneurysm may be excised after having taken down the previous anastomosis, and then the same graft may be reanastomosed to the well-preserved original recipient artery. In most instances, however, the changes in the recipient artery are so advanced that resuturing is not possible and the recipient artery and a segment of the prosthesis have to be excised. In these cases the area of origin of the deep femoral artery is used for an anastomosis. This type of repair also usually necessitates the ligation of external iliac artery.

INFECTION. As already mentioned, the overwhelming majority of infections of an arterial reconstructive operation begin at the time of the surgical intervention and are a result of direct invasion of the wound by bacteria. In current clinical practice, infectious complications occur almost exclusively after aortofemoral bypass operations. Infection of a prosthesis for the repair of visceral arteries or the carotid artery is exceedingly rare, as is also infection of a graft of autogenous

tissue origin. The discussion that follows, therefore, may conveniently be limited to the subject of aortic graft infections.

In an investigation of 40 patients with infected aortic prostheses we found that two-thirds of the infections originated in the groin and that in two-thirds of patients the offending organism was Staphylococcus aureus. There is strong clinical evidence that the appropriate use of prophylactic antibiotics is effective in reducing the possibility of aortic graft infection.

It should be emphasized that graft infections that originate in a groin wound rarely remain localized in that site. Most of these infectious lesions spread along the prosthesis proximally for varying distances, often extending as far as the opposite limb of the graft. This fact makes it necessary in most cases to remove the entire prosthesis with the insertion of an appropriate ex situ bypass graft.

A smaller but very important group of infectious complications results from the escape of intestinal contents at the prosthesis owing to a formation of fistula between a hollow viscus and the tissue environment of the prosthesis. In most of these cases the source of infection is the duodenum and communication is between the duodenum and either the tissues surrounding the graft or the lumen of the graft itself. Three types may readily be distinguished: aortoprosthetic fistula, aortoparaprosthetic fistula with low grade infection, and aortoparaprosthetic fistula with acute sepsis.

The pathologic features of these lesions determine their clinical manifestations and their management. In an aortoprosthetic fistula the communication is between a hollow viscus (almost always the duodenum), and the suture line, and the clinical manifestation is hemorrhage. Although at first bleeding from the upper gastrointestinal tract may be mild, soon there is massive blood loss. In a case of paraprosthetic fistula with low-grade infection the pathologic change consists of an erosion of the duodenum with an escape of the duodenal contents, creating a local cellulitis of low intensity that may involve varying portions of the shaft of the graft but does not reach the suture line. This distinction is important because without the involvement of the suture line bleeding does not take place. In these cases the clinical manifestations are those of low-grade infection and differential diagnosis may be extremely difficult. In aortoparaprosthetic fistulas with acute sepsis, the etiology is similar to that described for the previous lesion but a virulent infectious process extends from the site of origin along most or all of the implants and there are systemic septic manifestations (often with metastatic abscesses).

Treatment in every instance is surgical, although antibiotic therapy as an adjunct is of great importance. In patients with aortoprosthetic fistula with bleeding, surgical intervention must be prompt. In these patients it is virtually never possible to reconstruct the suture line, and it is necessary to suture ligate the aorta and remove the graft. The duodenal defect also must be repaired, and an ex situ bypass (usually an axillofemoral bypass) must be created to provide distal blood supply. In the aortoparaprosthetic fistulas in which the duodenum does not communicate with the aorta and which are not associated with septic manifestations, it is often possible to save the

prosthesis. After careful repair of the duodenum, the infected area is debrided and a pedicled omental graft is placed around the area of involvement of the shaft of the prosthesis. Irrigation with antibiotics through a small retroperitoneal catheter is optional. Appropriate postoperative antibiotic medication of massive doses is necessary. When the paraprosthetic infection, even though not involving the suture line, has caused acute sepsis, the salvage of the graft is not recommended. In these cases the treatment is as outlined for the aortoprosthetic fistula with bleeding.

Progression (Recurrence) of Atherosclerosis. Complications due to the progression or recurrence of atherosclerosis are best dealt with together since in many instances it is difficult or impossible to tell whether a given lesion that has led to complication is a recurrence or an advancing, more severe form of the original atherosclerosis. In general, when atherosclerotic late complications occur after an endarterectomy the cause is recurrence, whereas late occlusive complications with atherosclerosis after grafting procedures are as a rule a result of progression of the disease.

Regardless of the form of original surgical technique, whether grafting or endarterectomy, the affected segment of the arterial tree is likely to show recurrence or progression owing to the fundamental nature of atherosclerosis. There are no accurate statistical data to indicate how often this phenomenon is the cause of late failures in the various topographic regions of the arterial tree. We carried out a study of the problem on aortoiliac endarterectomies and found that even though in a high percentage of the arterial segments originally operated on the atherosclerosis showed some degree of recurrence, the reappearance of the occlusive process was usually mild, and during the first eight years of observation it rarely caused complete obliteration of the lumen.[7]

Atherosclerotic recurrence or progression is the most frequent cause of late occlusion of an aortofemoral bypass or replacement graft. The mechanism of occlusion is the reappearance of the atherosclerotic process in the common femoral artery at the site of bifurcation, usually primarily affecting the deep femoral artery (in most instances the superficial femoral artery having already been occluded at the original operation). In these patients the correction of the occlusion of the limb of the graft must always include a careful reconstruction of the endarterectomy in the area of the bifurcation of the common femoral artery. Whether the remainder of repair will consist of the insertion of another aortofemoral bypass (usually bilateral) or of an ex situ bypass, will depend on the type of operative risk the patient presents. In general, there is good evidence to show that an aortofemoral bypass has a greater probability for continued patency than an axillofemoral bypass and therefore, if risk factors allow, it should be preferred.

In this connection it should be mentioned that (in addition to structural change in the graft and recurrent atherosclerosis in the common femoral arteries) the third cause of the loss of patency of a limb of an aortofemoral bypass is thrombosis as a result of hypotension or dehydration in absence of significant atherosclerotic occurrence in the femoral area or of morphologic change in the graft proper. In these

cases the thrombus can be removed and patency restored through a simple procedure.

The recurrence of atherosclerotic changes causing occlusion after aortoiliac endarterectomy can be best handled by the insertion of an aortobifemoral Dacron bypass. As formerly mentioned in the management of late occlusion resulting from morphologic change in the aortofemoral graft, the complete removal of the affected atherosclerotic arterial segment is not necessary and the dissection can be limited to the areas of anastomosis.

DISCUSSION

The surgical management of complications following reconstructive arterial procedures requires not only a considerable degree of skill but often also a facility in improvisation. The technical challenge of correction is often great and puzzling. Frequently new technical details must be devised to solve the problem of a novel situation created by the complication. On the other hand, the results can be highly gratifying. A review of our own experience with 4247 reconstructive surgical procedures among which complications had to be treated with 557 secondary operations (13.1 per cent) showed a success rate ranging from 60 to 90 per cent, according to the type of complication but excluding the procedures used in the management of infection. (Only 40 per cent of the infected grafts could be managed without loss of life or limb.) The results unquestionably justify an aggressive surgical therapeutic attitude toward these lesions. The prerequisite of the full exploitation of the potential of such an attitude, in turn, is a careful and systematic follow-up routine for the observance of the stability of the primary postoperative results. Only such fastidious follow-up will detect early evidences of late complications and thus make it possible to correct a failure early in its development.

REFERENCES

1. Smith, R. F., and Szilagyi, D. E.: Ischemia of the colon as a complication in the surgery of the abdominal aorta. Arch. Surg., *80*:806, 1960.
2. Szilagyi, D. E., Elliott, J. P., Hageman, J. H., et al.: Biologic fate of autogenous vein implants as arterial substitutes: Clinical, angiographic and histopathologic observations in femoro-popliteal operations for atherosclerosis. Ann. Surg., *178*:232, 1973.
3. Szilagyi, D. E., Elliott, J. P., Smith, R. F., et al.: Secondary arterial repair: The management of late failures in reconstructive arterial surgery. Arch. Surg., *110*:485, 1975.
4. Szilagyi, D. E., Hageman, J. H., Smith, R. F., et al.: Spinal cord damage in surgery of the abdominal aorta. Surgery, *83*:38, 1978.
5. Szilagyi, D. E., Smith, R. F., Elliott, J. P., et al.: Anastomotic aneurysms after vascular reconstruction: Problems of incidence, etiology, and treatment. Surgery, *78*:800, 1975.
6. Szilagyi, D. E., Smith, R. F., Elliott, J. P., et al.: Infection in arterial reconstruction with synthetic grafts. Ann. Surg., *176*:321, 1972.
7. Szilagyi, D. E., Smith, R. F., Whitney, D. G.: The durability of aortoiliac endarteriectomy: A roentgenologic and pathologic study of late recurrence. Arch. Surg., *89*:827, 1964.

Department of Surgery
Henry Ford Hospital
2799 West Grand Boulevard
Detroit, Michigan 48202

Surgery of the Aortic Arch Branches and Vertebral Arteries

Edwin J. Wylie, M.D., and David J. Effeney, M.D.†*

This article describes the patterns of atherosclerotic lesions involving the proximal segments of the extracranial arteries supplying the brain, defines the indications for surgical intervention, and describes the surgical methods that have been found at the University of California, San Francisco, to provide the most satisfactory and durable results.

PATHOLOGIC PROCESSES

Stenosis

Atherosclerosis of the major brachiocephalic trunks tends to involve an anatomic zone that includes the dome of the aortic arch and the orifices of the three major branches. Often the lesion is a single large plaque in the aorta with overflow stenosis of all the branches. At other times the prominent and clinically significant area of intimal disease will be confined to the orifice of only one of the arteries.

The aortic portion of the lesion rarely involves more than one-third of the aortic circumference and has an easily palpable ledge at its inferior border. The lesion may extend proximally from the innominate orifice for 1 to 3 cm along the lateral wall but stops well distal to the pericardial reflection. When operation requiring a graft from the aorta is necessary, the portion proximal to this plaque is used for the origin of the graft.

The primary lesions in the three branches are in their proximal segments. In the left common carotid artery the disease tends to confine itself to the first two centimeters. In the innominate artery the atherosclerotic lesion often extends distally along the posterior and

*Professor of Surgery and Vice-Chairman, Department of Surgery, University of California, San Francisco; Chief, Vascular Surgery Service, University of California, San Francisco, California

†Assistant Professor of Surgery, University of California, San Francisco; Staff Vascular Surgeon, Veterans Administration Hospital, San Francisco, California

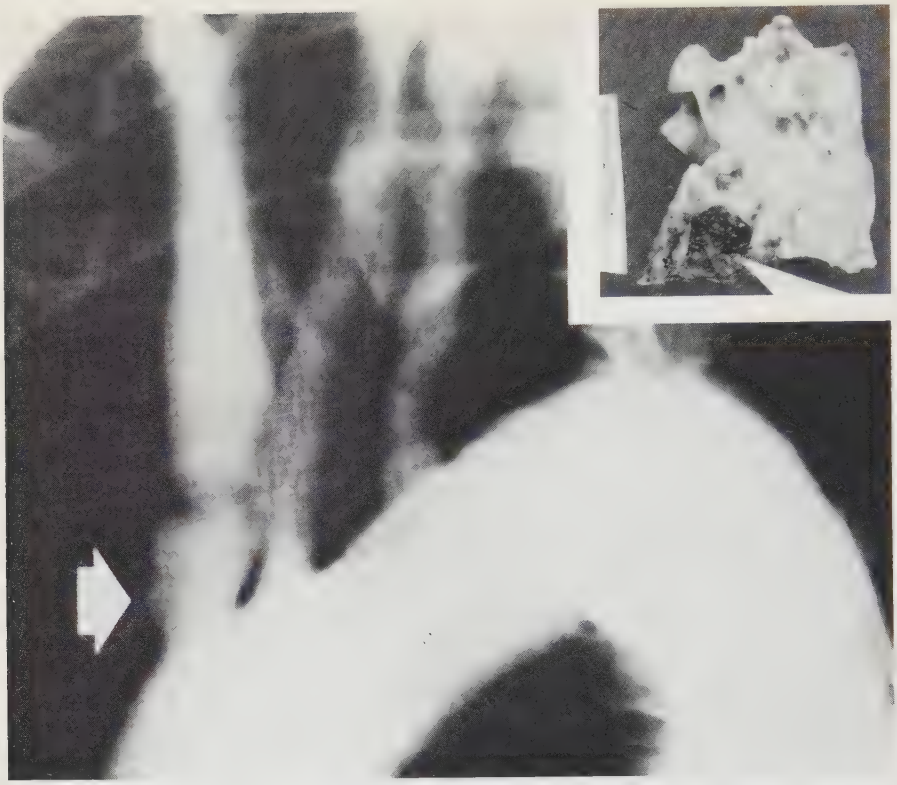


Figure 1. An arch aortogram shows the location of an ulcerating lesion (inset) in the innominate artery which had been the cause of transient ischemic attacks in the right hemisphere.

right lateral walls and even into the posterior wall of the right subclavian artery, but spares the intima at the right common carotid orifice. A similar pattern of distal progression is frequently encountered in the left subclavian artery. In many patients with vertebral stenosis, the vertebral lesion is an extension of the subclavian lesion proximal to its orifice.

Ulcerating lesions may appear in the wall of the aorta proximal to the innominate artery and in the proximal portions of both the innominate and left subclavian arteries and become the nidus for peripheral embolization to the brain or upper extremities. The innominate artery is the most common location of such ulcers (Fig. 1). We have never encountered ulceration in the proximal left common carotid artery.

An unusual variant that has received scant attention in the surgical literature may result from ulcers at these sites. A propagating mural thrombus may dangle into the aortic lumen from a lesion at the orifices of the innominate or left subclavian arteries and create major problems of distal embolization. The true source of such emboli may often escape detection.

Atheroscelerotic stenosis or occlusion of the right subclavian artery may occur in the absence of innominate disease and often appears as a curiously localized diaphragm of atherosclerotic thickening of the intima.

Atherosclerotic stenosis of the vertebral arteries is generally localized to their orifices and has a sharp end-point within 1 or 2 cm of the orifice. We have encountered only one patient with nonatherosclerotic kinking of the vertebral artery. External compression from fibrous bands has been reported but none has been observed in our own experience.

Patterns of Occlusion

When stenosis progresses to occlusion, as with atherosclerotic occlusion in other major arteries, the extent of occlusion is determined by the reentry point of collateral blood flow. Thus innominate occlusion extends only to its bifurcation, at which point carotid-to-subclavian flow maintains distal patency. Proximal subclavian occlusions tend to be limited to the reentry point of retrograde blood flow from the vertebral arteries.

When the vertebral arteries are occluded from progressive atherosclerosis at their orifices, thrombosis propagates distally to the C_2 level where blood returns from the distal vertebral muscular branches that have numerous connections with branches of the external carotid arteries. Since surgical access is easily obtained for the full length of innominate and subclavian occlusions, these lesions are amenable to direct techniques for reconstruction. Successful operations to bypass vertebral occlusions by grafts from the external carotid to the distal vertebral arteries have been reported, but these are difficult to perform and are generally reserved for patients with severe disability in whom no other reconstructive procedures are feasible.

When the common carotid arteries become occluded, the full length of the common carotid segment is involved. Extension of occlusion beyond the distal end of the common carotid arteries is determined by the pattern of atherosclerotic involvement at the carotid bifurcation. On the right side, the primary lesion is invariably at the bifurcation where atherosclerosis involves all three arterial components. If the common carotid component is the first to progress to total occlusion, distal patency is maintained by external-to-internal carotid blood flow. This collateral circuit to the ipsilateral hemisphere can be considered only temporary, since it remains vulnerable to eventual closure with the further progression of atherosclerosis. With occlusion, thrombus propagates distally in the internal carotid artery to its intracranial branches. In the external carotid artery, however, occlusion remains confined to its first 1 to 3 cm, because further extension of thrombus is halted by the inflow of blood from its terminal branches.

The patterns of progression of the disease have two aspects of clinical importance: (1) Although occlusion of the full length of the common carotid artery that extends only to its bifurcation may occur with few or no symptoms, progression of the distal lesion to occlusion will

substantially reduce blood flow to the cerebral hemisphere and often results in cerebral infarction. (2) Patients with occlusion of all three arterial components who have not suffered a stroke may often be disabled by hypoperfusion symptoms that may be relieved or lessened by an operation in which the common carotid artery and the occluded orifice of the external carotid artery are reopened to increase the collateral flow to the ipsilateral hemisphere.

The same considerations apply to the occlusion of the left common carotid artery if the primary lesion is confined to the area of bifurcation. Even when the occlusion results from closure of a proximal lesion adjacent to the aorta, clot propagates to the level of bifurcation and because of the tendency to continue to propagate creates the same threat to the patient.

MECHANISMS OF CEREBROVASCULAR INSUFFICIENCY

The collateral circulation around occlusions of the proximal brachiocephalic trunks is particularly abundant.[3] Thus, occlusion of any one of them rarely results in cerebral infarction. The only exception is occlusion of the left common carotid artery and even in this situation, infarction is rare unless the left internal carotid artery is also occluded in tandem.

A rich collateral supply is also available to the distal vertebral and the basilar arteries when stenosis or occlusion involves the proximal vertebral arteries or the adjacent subclavian arteries. We have encountered only one patient who suffered infarction in the posterior territory as a result of the bilateral progression to occlusion of the vertebral arteries.

The only other cause of cerebral infarction, embolization, is unrelated to collateral circulation, since it usually occurs from ulcerating lesions in the innominate artery.

The primary indication for operation is the disabling symptoms that may occur from generalized cerebral hypoperfusion. When these involve the carotid territory they present as postural giddiness, episodic unilateral dimming of vision, or more rarely as frank transient ischemic attacks. In the vertebral basilar territory they present as episodic binocular visual blurring, diplopia, ataxia or drop attacks. Postural giddiness, which appears to be a nonlocalizing symptom, is also frequent in patients with pure vertebral lesions. In symptomatic patients, multiple lesions are usually demonstrated. The only single lesions in which collateral blood supply is often inadequate to ward off such symptoms are advanced stenosis or occlusion involving the innominate or common carotid arteries. Symptoms of hypoperfusion in the vertebrobasilar territory from subclavian or vertebral lesions occur only when the involvement is bilateral or when associated lesions are present at one or both bifurcations of the common carotid artery.

A similar abundance of collateral blood supply is available to the upper extremities in patients with obstructive lesions in the proximal

subclavian arteries. Symptoms of ischemia are confined to exertional fatigue in the forearm that occurs only when extreme and prolonged exertion is required.

INDICATIONS FOR OPERATION

From the foregoing discussion, it may be assumed that the necessity for vascular reconstruction of these vessels is uncommon. Table 1 is a tabulation of our total surgical experience with extracranial occlusive cerebral vascular disease and shows the relative infrequency of operations performed for proximal aortic branch or vertebral disease, compared to distal carotid lesions. Many of the patients with carotid operations had associated lesions in one or more of the proximal cervical or mediastinal arteries. Less than 6 to 8 per cent of all patients with hemodynamically significant proximal lesions demonstrated by physical findings or arteriography required operations to remove or bypass these lesions. Eighty-five per cent of symptomatic patients with combined carotid and subclavian or carotid or vertebral lesions became asymptomatic after correction of the carotid lesion.

Embolization of the brain, the eye, or an extremity is a clear indication for operation. Symptoms of hypoperfusion are an indication only when the continuation of these symptoms is a genuine hazard to the patient or to others, such as in the patient unable to descend a staircase safely or cross a roadway or one who must operate a motor vehicle. The only exception to this statement occurs with common carotid occlusions where the presence of the lesion is in itself an indication for operation.

Any fatigue from nonembolizing subclavian lesions becomes an indication for operation only when genuine occupational disability results. Carpenters, mechanics, or house painters have occupations in which such disability could be a serious limitation. More severe degrees of ischemia in the upper extremities generally occur only when the subclavian lesion has produced many peripheral emboli. Another mechanism for embolization from subclavian arteries that must be

Table 1. *Sites of Operation for Atherosclerotic Extracranial Cerebrovascular Disease (1958–1978)*

SITE	OPERATIONS	
	Number	Per Cent
Carotid bifurcation	1769	90.4
Subclavian	84	4.3
Vertebral	42	2.1
Innominate	34	1.7
Common carotid	32	1.5
Total	1961	100.0

Table 2. *Operations Performed for Atherosclerosis of the Brachiocephalic Trunks and the Vertebral Arteries*

	INNOMINATE	COMMON CAROTID	SUBCLAVIAN	VERTEBRAL
Endarterectomy	28	15	49	36
Transposition		7	7	6
Endarterectomy plus Transposition		7	3	
Bypass Grafts from Aorta	5	2	1	
Transcervical	1		21	
Graft Replacement		1	3	
Total	34	32	84	42

recognized is the dislodgement of fragments of mural thrombus from the wall of a post-stenotic aneurysm beyond a compressing structure, usually a cervical rib, at the thoracic outlet. Surgical decompression and replacement of the arterial segment are required in these patients.

SURGICAL METHODS

The types of operations performed during our total experience from 1958 to 1978 are shown in Table 2. It is beyond the scope of this article to attempt to lead the reader through each of the steps in the evolution of the choice and application of techniques that are currently preferred and performed in our own clinic. As with the development of any other surgical methodology, prime reliance was placed upon evaluation of our own results. In this assessment, we considered the critical issues to be the safety of the technique, its effectiveness in the relief of symptoms, and its durability. Two other factors have influenced our current attitudes for the selection of the most appropriate operation: our concurrent experience with operations for revascularization in other portions of the arterial tree, and the inspection of surgical results by colleagues employing other techniques, with information obtained either by direct report or by examination of patients who had been referred to us after the original operation had failed. Certain guiding principles have evolved.

(1) Operations which deal directly with the arterial lesion, whether endarterectomy, graft replacement, or "in line" bypass grafts, are generally preferable to the more remote transcervical bypass operations.

(2) Endarterectomy is the preferred direct operation for nonaneurysmal occlusive lesions, provided safe access to the diseased segment can be obtained.

(3) As a corollary to (1) and (2), approach to the innominate or the proximal left subclavian arteries by a median sternotomy or a lateral thoracotomy incision provides what now can be considered a safe access to these arteries for performing a direct operation.

(4) When bypass operations are used, they must exclude from circulation any lesion which has been, or may become, a source of cerebral embolization.

The remainder of this article deals with aspects of specific operations performed in our own clinic in the past two decades for lesions at the various sites described above. For a description of the details of operative technique, the reader is referred to *A Manual of Vascular Surgery* by Wylie et al., and to three previous publications from our vascular unit.[1, 2, 4]

Innominate Artery

A direct operation within the mediastinum was performed in all but one of 34 patients. Access was obtained through a median sternotomy approach. In the operations early in our experience the sternotomy was terminated by a hockey stick extension into the right fifth interspace. We currently employ a full-length sternotomy with extension of the incision upward along the medial border of the right sternocleidomastoid muscle.

Endarterectomy was performed whenever a partially occluding aortic clamp could be applied to encircle the aortic portion of the lesion without encroaching upon it (28 patients). The distal end-point of the endarterectomy was in the innominate artery in 20 patients; in eight extension of the endarterectomy into the right subclavian artery to reach a satisfactory end-point was necessary.

Bypass of the innominate artery with a Dacron graft originating from the side of the ascending aorta was necessary in five patients. In these patients the dome of the arch adjacent to the innominate artery was so widely involved that it was considered hazardous to apply a partially occluding aortic clamp for the performance of innominate endarterectomy. Two patients had associated tight stenotic lesions at the left common carotid orifices and in these, innominate endarterectomy was combined with an aorta-to-common carotid graft also originating from the side of the ascending aorta (Fig. 2).

A transcervical subclavian-to-subclavian graft of a saphenous vein segment was used in one patient who had undergone a previous incomplete endarterectomy at another hospital.

The presenting symptoms were those of chronic cerebral hypoperfusion in 27 patients. Six patients had experienced transient ischemic attacks in the right hemisphere or amaurosis fugax in the right eye from ulcerating lesions at the orifice of the innominate artery. All patients have been relieved of their symptoms without recurrence except for the one patient with a transcervical graft which subsequently closed.

Subclavian Artery

Endarterectomy. Endarterectomy was used in all patients with atherosclerotic stenosis of the proximal right subclavian artery (40) and in 14 of 43 patients with proximal lesions of the left subclavian artery. Operations on the right side were performed through a cervical

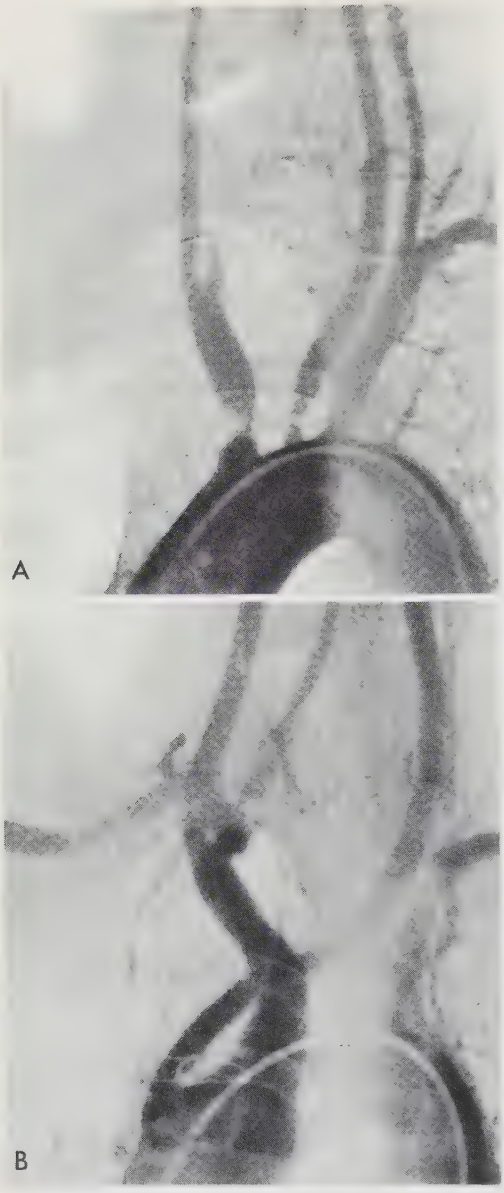


Figure 2. Preoperative (*A*) and postoperative (*B*) arch aortograms show atherosclerotic stenosis of all three brachiocephalic trunks, most severe in the innominate and left common carotid arteries. The innominate artery was opened by endarterectomy. A 6 mm Dacron bypass graft was placed from the side of the ascending aorta to the transected distal end of the left common carotid artery.

incision and on the left through a third interspace left thoracotomy incision. Two patients died early in our experience after an endarterectomy for a left subclavian occlusion. Both deaths were preventable. One occurred from cardiac arrest following anesthetic difficulty and one from stroke as a result of unobserved inclusion of the left common carotid artery in the partially occluding aortic clamp. Associated lesions of the vertebral orifice were removed with the subclavian speci-

men in seven patients. There have been no early or late closures of the endarterectomized segments.

Transposition. The left subclavian artery was transposed to the side of the left common carotid artery on the left side in 10 patients, all but one through a cervical approach with the anastomosis at the level of the clavicle. An eversion endarterectomy of the distal subclavian stump was required in three patients. The successful performance of transposition requires careful mobilization of the proximal subclavian artery and transection midway between its orifice and the orifice of the left vertebral artery. Transposition is limited to patients with normal patency of the vertebral orifice. There were no postoperative complications or late closures.

Both endarterectomy and transposition operations comply with most of the guidelines stated previously and neither requires the interposition of grafts. The transposition operation is useful for the occasional patient in whom a thoracotomy may be inadvisable. When the proximal anastomosis is placed deep in the neck, there is minimal deviation of the direction of flow. Both operations have demonstrated their durability. All patients have been relieved of their symptoms.

Aorta-subclavian Graft. In one patient a Dacron bypass graft was installed from the descending thoracic aorta to the transected distal end of the left subclavian artery in a patient with multiple emboli to the arm from a lesion at the orifice. This occluded at 24 hours. The Dacron was removed and the subclavian artery was transposed to the side of the left common artery from within the chest. We suspect that the acute reversal of the direction of flow in the original graft was a factor contributing to its early occlusion.

Transcervical Bypass Graft. There were 21 bypass grafts, all on the left side and all from the left common carotid artery to the left subclavian artery. The saphenous vein was used in 17 and a Dacron graft in four. There were no immediate failures. Five of the saphenous veins have closed in periods from four to eight years postoperatively.

Replacement Grafts. In three patients replacement grafts were used after resection of post-stenotic subclavian aneurysms that had been the source of emboli to the arm. One was a Dacron graft and two were arterial autografts. All have been functioning satisfactorily for periods of as long as 15 years.

All of the patients who had subclavian operations for cerebrovascular symptoms had either subclavian or vertebral lesions on the opposite side. The contralateral lesion was atherosclerosis in the subclavian or vertebral arteries or hypoplasia of the vertebral artery. All obtained symptomatic relief from a unilateral operation. None required an operation on the opposite side.

Common Carotid Artery

Total occlusion of the right common carotid artery was corrected by endarterectomy in 15 patients using an open technique at the distal end and an arterial stripper for the remainder. The primary lesion in each was an atherosclerotic occlusion at the distal end with subsequent proximal propagation of thrombus. The right common carotid

artery in one patient with postradiation atherosclerosis and fibrosis was resected and replaced with an arterial autograft taken from the right external iliac artery.

Seven patients had stenosis at the aortic orifice of the left common carotid artery. In four, the left subclavian artery was undiseased, permitting transcervical transfer of the carotid artery to the subclavian artery. In two patients, mentioned previously, extensive disease was present in all three brachiocephalic branches. Simple innominate endarterectomy was combined with antegrade Dacron bypass graft from the side of the ascending aorta to the end of the transected mediastinal portion of the left common carotid artery. Both patients became free of symptoms even though residual lesions in the left subclavian arteries remained.

Seven patients had total occlusion of the full length of the left common carotid artery. The primary atherosclerotic lesions were at the distal end. The left subclavian arteries were undiseased. The common carotid artery was opened by endarterectomy using the ring stripper and transposed to the left subclavian artery through a cervical approach.

Occlusion of the ipsilateral internal carotid artery was present in six of the 22 patients with total occlusion of one or the other common carotid arteries. Operation was performed to restore forward flow into the external carotid artery with the objective of improving collateral blood supply to the ipsilateral cerebral hemisphere. In two patients this operation was performed to prepare the way for a subsequent superficial temporal-intracranial bypass operation.

Patency was restored in all of the operations on the common carotid artery. There were no complications and no late closures.

Vertebral Artery

Revascularization operations for stenosis of the vertebral artery were performed in 42 patients. Seventy per cent of the lesions were confined to the vertebral arteries bilaterally. In 30 per cent there were associated lesions in the adjacent subclavian arteries. Operation was limited to one side in all patients. In patients with bilateral stenosis of the vertebral orifice, if the vertebral arteries were of comparable caliber with equal inflow, the right side was chosen for operation because it is simpler of access.

RIGHT VERTEBRAL ARTERY. Access to the right vertebral artery was by a supraclavicular cervical approach in 20 patients and was supplemented by a median sternotomy in two. Resection of the medial head of the clavicle, used in four of our early operations, has been abandoned because of the functional and cosmetic deformity it causes.

Endarterectomy of the vertebral orifice through a longitudinal subclavian arteriotomy was the only procedure used. The vertebral lesion had a palpable abrupt end-point in all patients. (In two, not included in this series, diffuse and lengthy atherosclerosis was palpated and operation was not attempted.) A patch graft was used in only one patient, since the resulting vertebral lumen of almost all patients becomes larger than normal after endarterectomy in its orifice.

LEFT VERTEBRAL ARTERY. A similar technique was used for atherosclerotic stenosis of the left vertebral artery in 14 patients. The lower level of the vertebral origin, however, permitted the cervical approach in only six patients. In the other eight a transthoracic approach was used. In the total of 36 endarterectomy operations for vertebral stenosis, associated subclavian lesions were removed in six.

Transposition of the left vertebral artery to the left common carotid artery (3) and to another position on the left subclavian artery (3) were performed in six patients with deeply situated vertebral arteries who were not candidates for thoracotomy.

There was one postoperative stroke among the patients with vertebral operations. This patient had an associated occlusion of the left internal carotid artery as well as bilateral vertebral lesions. A left hemispheric infarct, which led to death, was believed to be the result of the temporary interruption of collateral blood supply from the vertebral artery to the involved hemisphere. Follow-up information has been available on 37 patients. Symptoms in all but 7 per cent (3 patients) have been relieved or lessened. Arteriograms in the three failures showed occlusion of the endarterectomized vertebral artery in one. Although our arteriographic follow-up is incomplete, this is the only known postoperative occlusion.

COMMENTS

During the 20 years in which the various procedures described in this series were being used at our institution, other surgeons have more extensively employed one or more of the transcervical bypass operations for carotid, subclavian, and innominate lesions. The advocates of these operations cite the ease of performance and the usually satisfactory immediate result. Our own experience with these techniques supports these contentions. The durability of these operations, however, is open to question. Published data in this regard is scanty. Our own experience is limited to patients with unilateral carotid-subclavian grafts, usually employing saphenous vein segments. The late failure of these grafts now approaches 25 per cent, an attrition rate comparable to that occurring when the saphenous vein is used in other portions of the arterial system. Short bypass grafts of Dacron fabric are intrinsically less vulnerable to late occlusion but introduce a new threat should infection develop in the wound.

More extensive cervical crossover grafts using the subclavian or even the axillary arteries as donor or recipient vessels create new problems. The 180° redirection of blood flow at one or both ends creates zones of turbulence conducive to thrombosis. Grafts that pass interior to the trachea or the sternum are unsightly and susceptible to thrombosis from external compression.

It has been our experience that the direct operations, some of which require entering the anterior mediastinum or the left hemithorax, can be performed with equal ease and with an acceptable morbidity and permit the more frequent use of endarterectomy. This autogenous tissue technique preserves arterial integrity in the presence

of infection, has proved durable and causes the least redirection of blood flow. Arterial transposition operations avoid the introduction of foreign graft material and have been free of late failure. If synthetic grafts are necessary, as is occasionally the case, they can be used to replace the diseased segment or even to provide an "in-line" bypass graft with minimal redirection of blood flow.

We have restricted the use of synthetic grafts to patients in whom endarterectomy was considered too hazardous and then only when the graft would bypass or replace large-caliber arteries and when experience elsewhere has shown their success rate to be the highest. Whenever possible antegrade "in-line" positioning of the graft has been used to minimize turbulence of the anastomotic sites.

SUMMARY

Experience with 192 operations of vascular reconstruction for atherosclerosis in the proximal brachiocephalic and vertebral arteries is reported. These procedures constitute only 10 per cent of operations for extracranial arterial occlusive cerebrovascular disease at the University of California, San Francisco, in the past 20 years. All patients were asymptomatic. Except for six patients with cerebral embolization from ulcerating lesions, symptoms resulted from cerebral hypoperfusion. Prevention of ultimate stroke was the primary objective of operation in patients with embolization and in patients with stenosis or occlusion of the common carotid arteries. Purely obstructive lesions in the subclavian and vertebral arteries were symptomatic only when there was bilateral involvement and the objective of operation was the relief of disabling symptoms of hypoperfusion for these otherwise essentially benign lesions. Prior correction of associated stenosis of the carotid artery often removed the need for a proximal operation.

The majority of the operations were endarterectomy or transposition, or combinations of the two. Cervical bypass grafts, because of their less certain durability, were used only when a more direct operation was neither feasible nor safe.

REFERENCES

1. Carlson, R. E., Ehrenfeld, W. K., Stoney, R. J., et al.: Innominate artery endarterectomy — A 16 year experience. Arch. Surg., *112*:1389, 1977.
2. Ehrenfeld, W. K. Chapman, R. D., and Wylie, E. J.: Management of occlusive lesions of the branches of the aortic each. Am. J Surg., *118*:236, 1969.
3. Newton, T. H., and Wylie, E. J.: Collateral circulation associated with occlusion of the proximal subclavian and innominate arteries. Am. J. Roentgenol., *119*:394, 1964.
4. Wylie, E. J., and Ehrenfeld, W. K.: Extracranial Occlusive Cerebrovascular Disease. Diagnosis and Management. Philadelphia, W. B. Saunders Co., 1970.
5. Wylie, E. J., Ehrenfeld, W. K., and Stoney, R. J.: A Manual of Vascular Surgery. New York, Springer, 1979.

Department of Surgery
University of California
350 Parnassus Avenue
San Francisco, California 94117

The Role of Profundaplasty in Revascularization of the Lower Extremities

*Victor M. Bernhard, M.D.**

The importance of the profunda femoris artery in the vascular supply of the ischemic lower extremity was first recognized by Morris and his associates[18] and by Leeds and Gilfillan in 1961.[11] Their clinical experiences demonstrated that the profunda femoris artery is capable of compensating for occlusion of the parallel superficial femoral channel. Subsequent reports of revascularization of the profunda femoris artery have consistently supported this thesis.[4, 7, 8, 10, 15, 16, 22]

ANATOMIC AND PHYSIOLOGIC BASIS

The profunda femoris artery, under normal circumstances, is the smaller of the two primary divisions of the common femoral artery. Its multiple branches not only distribute blood supply to the thigh, but also provide a series of arcades which link the hypogastric and lumbar branches of the aorta and iliac arteries with the genicular and recurrent branches of the popliteal and tibial vessels.[16] This normal anatomical arrangement provides the basis for a rich collateral system which can bypass many levels of obstruction in the iliac, femoral and popliteal trunks (Fig. 1). As a consequence of progressive obliteration of the superficial femoral artery by atherosclerosis, these alternate channels enlarge to serve the dual role of local distribution to the thigh and conduction of arterial flow to the calf and foot. Waibel and Wolff noted that the pressure in the popliteal artery measured during surgery in patients with superficial femoral occlusion, was further reduced by temporarily clamping the profunda femoris artery.[23]

Operative flow studies further support the thesis that the collateral function of the profunda femoris artery can effectively compensate for loss of flow through the superficial femoral artery. Flow into the lower

*Professor of Surgery, Medical College of Wisconsin, Milwaukee, Wisconsin

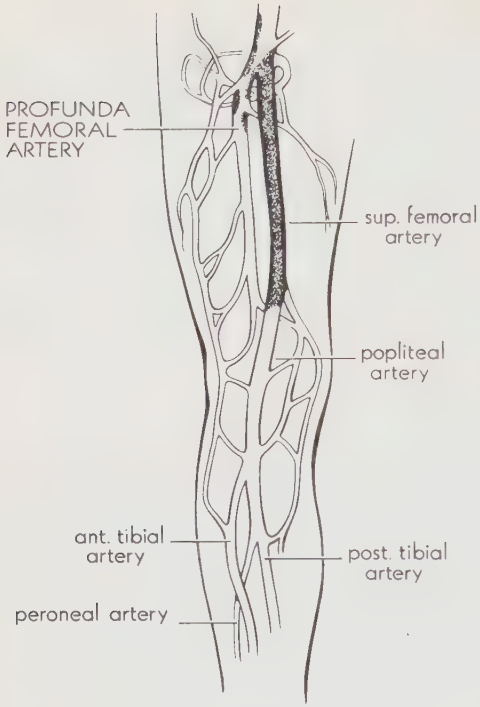


Figure 1. The anatomic relations and collaterals of the profunda femoris artery in the presence of occlusion of the superficial femoral artery. When atherosclerotic disease involves the deep femoral artery, it is generally confined to the orifice and proximal few centimeters of the vessel. (*From* Bernhard, V. M., Militello, J. M., and Geringer, A. M.: Repair of the profunda femoris artery. Am. J. Surg., *127*: 676, 1974, with permission.)

extremity through the profunda femoris artery following profundaplasty in patients with superficial femoral occlusion was demonstrated to be equal to flow through the external iliac artery of patients in whom both the profunda femoris and superficial femoral arteries were patent.[3] The volume of deep femoral flow was twice that routinely measured through femoral-popliteal bypass grafts.[3] Peripheral vasodilatation produced by the local intra-arterial injection of papaverine produces a significant increase in flow indicating that the arterial system of the profunda femoris vessel has the capacity to meet the demands of physiologic stress such as exercise.[3, 5] Postoperative increases in ankle pressures,[9] treadmill tolerance,[9] radionuclide blood flow,[22] and the volume of blood[14] further emphasize the importance of the profunda femoris artery in the circulatory economy of the ischemic extremity.

Nevertheless, resistance through the profunda femoris artery and its collateral branches even after maximal stimulus to hypertrophy is always greater than resistance through the normally patent superficial femoral artery. Strandness, et al. demonstrated objective improvement in tolerance for walking in only one-half of the patients subjected to treadmill exercise following profundaplasty.[21]

Berguer and his associates proposed, on the basis of geometric modeling, that the normal orifice of the profunda femoris artery produces a functional stenosis greater than 50 per cent when the superficial femoral artery is occluded.[3] The presence of only minimal athero-

sclerosis at this orifice significantly reduces the lumen and produces a significant obstruction to flow. On this basis, they and others recommend profundaplasty whenever the superficial femoral artery is occluded even in the absence of significant atherosclerotic obstruction of the orifice of the deep femoral artery.[19] Their hypothesis is supported by flow studies before and after profundaplasty which demonstrated that mean flow after papaverine vasodilatation increased significantly with widening of the orifice of the profunda femoris artery when only minimal disease was present.[3] Although this concept is intriguing, a larger body of clinical material must be carefully studied before accepting the concept that the orifice of the profunda femoris artery should be widened routinely in the presence of minimal stenosis in all ischemic limbs with superficial femoral occlusion.

PATHOLOGY OF ATHEROSCLEROSIS IN THE PROFUNDA FEMORIS ARTERY

The angiographic study of atherosclerosis in the lower extremities by Lindbom suggested that the profunda femoris artery was infrequently involved.[12] However, subsequent investigations utilizing oblique angiography confirmed by direct surgical observation demonstrated that obstruction of the profunda femoris artery was far more common than was originally reported.[2] Fortunately, when atherosclerosis involves the profunda femoris artery, it is generally localized to the proximal portion of the vessel in 74 per cent of the limbs. This conforms to the pattern noted in arteries of distribution and in collaterals where the main channel itself is normal and only the orifices are stenosed by intimal disease arising in the parent artery. Diffuse involvement of the profunda femoris artery or disease limited to its distal runoff portion is relatively infrequent. The angiographic investigation performed by Beales, et al. on 209 limbs demonstrated atherosclerotic involvement of the profunda femoris artery in 39 per cent.[2] Superficial femoral occlusion associated with some degree of involvement of the profunda femoris artery was noted by 21.5 per cent. However, severe stenosis of the profunda femoris artery (greater than 50 per cent) associated with superficial femoral obstruction was identified in only 7 per cent of the limbs studied. This information suggests the frequency with which one may anticipate that repair of the profunda femoris artery, either by itself or in association with an inflow procedure, may be useful in limb revascularization.

INDICATIONS FOR PROFUNDAPLASTY

Stenosis or occlusion of the deep femoral artery should be repaired whenever the distal portion of the artery is patent so that it may serve as the primary collateral to bypass an obstructed superficial femoral artery.

Profundaplasty is most frequently required as an adjunct to an inflow procedure such as aorto-femoral, axillo-femoral, or femoral-femoral bypass. This accounted for 58 per cent of the profundaplasties in our experience.[5] Failure to include this step in the initial procedure may lead to early thrombosis of a limb of an aorto-femoral graft as a result of unrelieved obstruction at the orifice of the profunda femoris artery. Late thrombosis of a limb of an aorto-femoral graft is usually caused by progressive atherosclerosis in this runoff vessel and profundaplasty is, therefore, an essential part of any operative procedure employed to revascularize a limb under these circumstances.[4]

Profundaplasty was performed as the primary operation without an inflow procedure in 23 per cent of our patients. This technique may be considered as an alternative to femoral-popliteal bypass when either option is available to the surgeon. However, stenosis of the profunda femoris artery should always be relieved before attempting tibial bypass. Finally, profundaplasty may provide the only potential for revascularization of a limb when neither the popliteal trunk or the tibial arteries are suitable for bypass. Primary repair of the deep femoral artery has the advantage of being a limited and minimally traumatic procedure which can be carried out through a simple incision in the groin. Furthermore, it does not risk injury to genicular or tibial collaterals which may occur at the time of bypass to the knee or calf and which may jeopardize the healing of a below knee amputation if that should subsequently become necessary.

Repair of a significant stenosis of the profunda femoris artery should be performed to insure healing of the stump below the knee whenever major amputation is inevitable and the blood supply at site of amputation is marginal. Doppler ankle pressures[1] or Xenon 133 flow studies[20] may serve as a guide to identify patients who will require revascularization under these circumstances.

SELECTION OF PATIENTS

Clinical findings alone are generally of little value in identifying those patients who will be candidates for reconstruction of the profunda femoris artery. However, the combined findings of a full femoral pulse, a femoral bruit, and demonstration of a depressed segmental pressure in the upper thigh strongly suggest that both the superficial femoral and the profunda femoris arteries are obstructed.

Angiography is the most effective technique for identifying patients in need of profundaplasty. Since the profunda femoris artery usually arises posteriorly from the common femoral artery, its orifice will be obscured by dye in the main trunk in the antero-posterior projection (Fig. 2).[2] Furthermore, the plaque which is frequently present in the common femoral artery and extends into the profunda femoris artery is almost invariably located on the posterior wall and will go unnoticed unless the lesion is seen in profile. The oblique projection

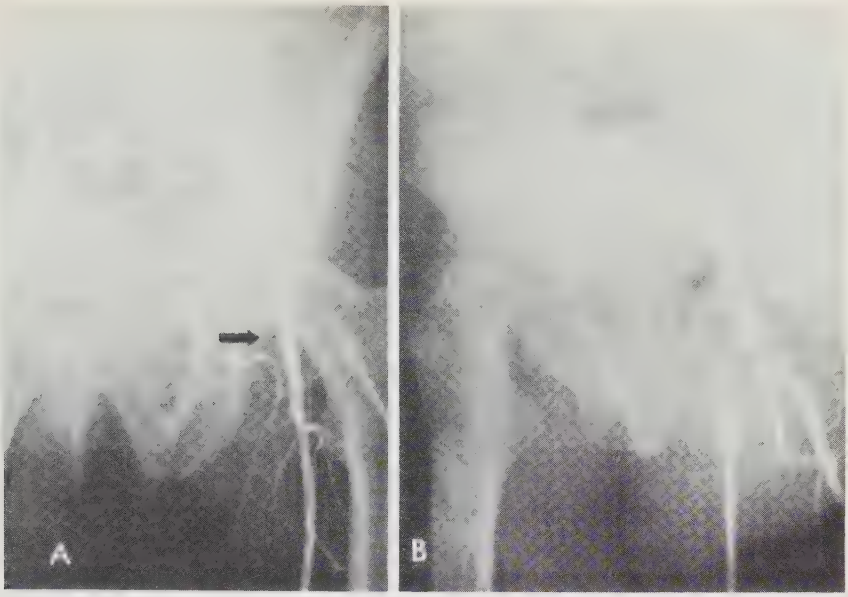


Figure 2. *A*, An arteriogram of the common femoral artery shows no apparent obstruction to flow into the profunda femoris artery on the anteroposterior view. *B*, The oblique projection reveals a severe obstruction in the proximal portion of the profunda femoris artery.

described by Beales and his associates should be used routinely to adequately display the proximal profunda femoris artery.[2]

Pressure measurement by direct needle puncture of the common femoral artery should be obtained at the time of angiography or during surgical exposure in order to rule out an occult but dynamically significant obstruction in the aortoiliac system. A pressure gradient, which is enhanced by the local instillation of 10 mg of papaverine to induce peripheral vasodilatation indicates the necessity for an inflow procedure in addition to repair of the profunda femoris artery.

Finally, the orifice of the profunda femoris artery should always be inspected and gently probed to determine that there is no significant obstruction whenever a common femoral arteriotomy is performed in preparation for anastomosis of a graft to the artery.

OPERATIVE MANAGEMENT

General anesthesia with full cardiovascular monitoring is recommended in most cases. However, when only a limited procedure through an incision in the groin is required, local anesthesia may be used.

The initial step in the procedure is to expose the femoral vessels. Exposure of the aorta, or some other source of arterial inflow, is delayed until after the repair of the profunda femoris artery is completed. A vertical incision which extends 5 cm proximal to the inguinal crease

is made directly over the femoral pulse or the hard cord of the occluded femoral artery. The incision runs parallel to the medial border of the sartorius as far distally as necessary to provide easy access to the diseased segment of the profunda. The incision should be deepened lateral to the lymph node chain in the groin, which is left intact and retracted medially in order to reduce bacterial contamination, avoid lymphorrhea, and minimize postoperative lymphedema. Injury to the femoral nerve and its branches which lie in the lateral aspect of the operative field, should be carefully avoided by meticulous dissection and judicious placement of retractors. The inguinal ligament is routinely divided to allow exposure of the common femoral artery at its junction with the external iliac artery. The occluded superficial femoral artery, which will subsequently serve as a source of autogenous patch material, is dissected free for a distance of several centimeters to facilitate identification of the deeper structures.

Exposure of the profunda femoris artery beyond the first two centimeters requires division of the large lateral circumflex femoral vein which lies transversely across the anterior surface of the artery (Fig. 3). It is a branch of either the common femoral artery or of the deep femoral vein. Dissection of the profunda femoris artery must be carried distally beyond obvious occlusive disease as determined by external inspection and palpation and reference to the arteriogram. Beyond

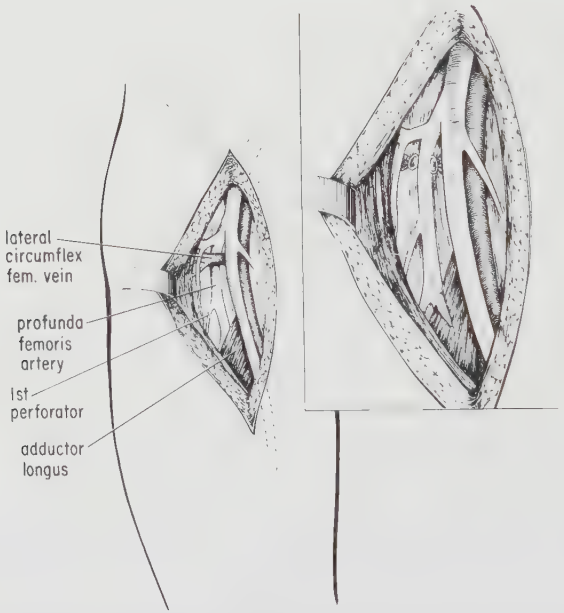


Figure 3. Surgical exposure of the proximal one-half of the deep femoral artery reveals the large lateral circumflex femoral vein arising from the common femoral vein. This vessel is located anterior to the main trunk of the deep femoral artery and must be divided in order to expose the deep femoral artery adequately beyond it.

this point, the vessel is usually soft and supple and the intima is relatively normal. The adductor longus muscle overlies the distal profunda femoris artery and must be incised to approach the artery beyond the second perforator. Considerable variation of the standard anatomy must be anticipated since the large lateral and medial circumflex femoral branches may arise from the common femoral or superficial femoral arteries rather than the profunda femoris artery. Therefore, all branches of these main trunks should be preserved.

An alternate approach to the deep femoral artery may be required when it is the only available runoff in a patient with an infected groin wound from a previous vascular repair. The old wound is isolated from the operative field by adhesive plastic drape. An incision is made parallel to the lateral border of the rectus femoris muscle at the junction of the upper and middle thirds of the thigh. The midportion of the profunda is demonstrated in the depths of a tissue plane between the rectus femoris muscle and the vastus medialis muscle. At this point, the vessel is sufficiently remote from the previous groin wound to insure a clean field.

The pressure in the common femoral artery is measured by direct needle puncture at this time if there is any doubt regarding the adequacy of the iliac inflow.[17] This pressure is compared with that obtained from an inlying radial arterial cannula. Peripheral vasodilatation is produced by injection of 15 mg of papaverine into the femoral artery in an attempt to unmask a pressure gradient due to an occult but dynamically significant iliac stenosis. A baseline gradient of 15 mm Hg between arm and femoral pressure which increases to 30 mm Hg after papaverine indicates the need for an inflow procedure in addition to the profundaplasty.

The patient is anticoagulated either systemically with 100 units of heparin per kilogram or by local installation of heparin into the distal vessel after arteriotomy. The common femoral and main profunda femoris vessels are occluded with soft, rubber jaw vascular clamps and the smaller branches are controlled with Heifetz microsurgical clamps or with slings of heavy silk. An arteriotomy is begun in the common femoral artery and extended into the orifice of the profunda femoris artery down to a point several millimeters beyond the major obstructing disease (see Fig. 3). At this point, the intimal lining of the vessel wall becomes relatively normal. An endarterectomy is performed in the plane of easy separation in the mid-media of the vessel. If a clean break cannot be achieved distally, fine tacking sutures are placed to stabilize the distal intima. Obstructing atherosclerotic plugs are removed from the orifices of branches by simple avulsion, since the disease usually extends only one to three mm beyond the junction of the branches with the main channel. These vessels are important contributors to the collateral function of the arterial system of the profunda femoris. Special attention should be paid to the large lateral circumflex femoral artery and its descending branch which may require a separate angioplasty (see Fig. 5B).

A tapered patch is routinely sutured over the arteriotomy to further widen the lumen. Malone and associates have demonstrated that autogenous tissue is distinctly superior to plastic patch materials.[13] Their findings concur with our experience. Although saphenous vein is the ideal patch material, suitable veins may not be present or should be spared for some future revascularization procedure. However, an endarterectomized segment of adjacent occluded superficial femoral artery provides an excellent source of autogenous material and is consistently available (Fig. 4). We make an exception to the use of an autogenous tissue patch when an inflow procedure is performed in conjunction with a profundaplasty which will have to extend no more than 1 to 2 cm into the deep femoral vessel. An extended tongue of the Dacron tube is the appropriate substitute in this circumstance. Occasionally, a simple patch without endarterectomy is performed to widen the lumen when the obstructing plaque is not ulcerated and is firmly applied to the vessel wall.

After the profundaplasty has been completed in patients who have aortoiliac obstruction, the inflow source vessel is exposed. The bypass graft is sutured to the aorta, opposite femoral or the axillary artery and tunneled to the groin. A 2 cm incision is made into the proximal end of the patch for end-to-side anastomosis to the prosthesis.

An intraoperative arteriogram is performed to outline the area of endarterectomy and patch angioplasty whenever the profundaplasty extends as far as the second or third perforator or the distal runoff channel is less than 3 mm in diameter (Fig. 5).

Postoperatively, the degree of improvement in distal perfusion is monitored by clinical examination, estimation of the quality of flow in the pedal vessels by Doppler ultrasound, and repeated measurement of ankle pressures. A weak pedal pulse occasionally reappears when the

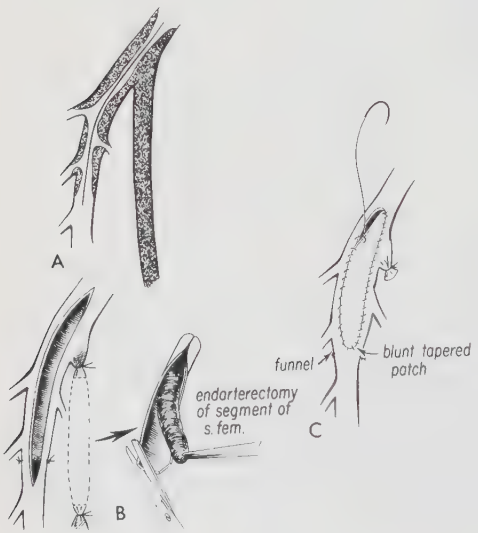


Figure 4. The technique of profunda femoris endarterectomy and patch angioplasty makes use of an endarterectomized segment of the occluded superficial femoral artery. (*From* Bernhard, V. M.: Profundaplasty. *In* Rutherford, R. B. (ed.): Vascular Surgery. Philadelphia, W. B. Saunders Co., 1977, ch. 59.)

Figure 5. *A*, Severe stenosis of the deep femoral artery is associated with partial obstruction at the common iliac level. *B*, A postoperative arteriogram demonstrates the completed profundaplasty with femoral-femoral bypass to provide satisfactory inflow. The patch angioplasty has been extended onto the lateral femoral circumflex artery.

popliteal and tibial vessels beyond the obstructed superficial femoral artery are patent. If circulation does not improve, an arteriogram should be obtained without delay to look for technical errors and to determine whether an adequate profundaplasty has been accomplished even though a good femoral pulse is palpable. Prompt reoperation should be performed whenever postoperative thrombosis develops or if a significant defect is identified on the arteriogram.

RESULTS

Claudication is relieved to some degree in almost all patients operated upon for this indication alone.[5, 8, 9, 23] However, complete relief is achieved in only 50 per cent of patients[8] because the collateral sys-

tem of the profunda femoris artery, even when fully developed, imposes a higher resistance to flow than does that of the normal superficial femoral artery.[9, 22] In our series, 95 per cent of patients were subjectively improved.[5]

The degree of improvement in limb perfusion is directly proportional to the patency of the popliteal and tibial vessels. In those patients operated upon for pain at rest, ischemic ulceration, and limited necrotic lesions of the foot and toes, limb salvage was always achieved when the popliteal artery and two or more tibial vessels were patent to the ankle. Success was achieved in 85 per cent of those patients with moderate disease of the popliteal and tibial outflow. However, when the popliteal artery was occluded and filling of the tibial vessels was meager, major amputation was avoided in only 46 per cent. Overall, salvage was achieved in 67 per cent of limbs subjected to profundaplasty and followed for five years by life-table analysis.[5]

Patients who require an inflow procedure in addition to profundaplasty generally have a better result than those who are subjected to profundaplasty alone. This is probably due to the status of the outflow at the popliteal-tibial level which is the ultimate factor limiting success of profundaplasty.[5, 22] Obstruction in the runoff vessels in the calf and foot is generally less severe in the limbs with combined obstruction of inflow, superficial femoral occlusion, and stenosis of the profunda femoris artery in comparison with limbs affected by a comparable degree of ischemia but with no inflow disease.

There was no operative mortality when operation was performed for claudication alone. This reflects our conservative approach to patients with functional ischemia for whom surgery is not recommended unless the operative risk is minimal. On the other hand, revascularization is readily offered to patients at greater risk when the only alternative is major amputation, which correlated with a 7 per cent mortality in patients operated upon for limb salvage in our series.

CONCLUSION

The deep femoral artery becomes the most important collateral vessel in the ischemic lower extremity when the superficial femoral artery is occluded. Profundaplasty is an important adjunct to any inflow procedure when the proximal portion of the deep femoral artery is stenotic, since it insures optimal restoration of circulation to the extremity and prevents early occlusion of the graft.

Repair of the profunda femoris artery is a reasonable alternative to femoral-popliteal bypass when either option is available to the surgeon and when narrowing of the profunda femoris artery is greater than 50 per cent. Stenosis of the profunda femoris artery should always be relieved before performing a tibial bypass. The former is simpler, less hazardous and frequently provides enough circulatory improvement so that the latter will not be necessary.

Precise angiography is the most effective means of selecting patients for profundaplasty.

Endarterectomy and patch angioplasty with autogenous tissue is the procedure of choice for repair of the profunda femoris artery and should be carried as far distally as necessary, even beyond the third perforator, to insure effective perfusion of all the collateral branches and revascularization of the limb. Improvement is directly proportional to patency of the popliteal and calf vessels.

Patients should be observed carefully in the immediate postoperative period and periodically thereafter with the Doppler velocity flowmeter and by repeated measurement of pressure at the ankle. Failure to achieve significant improvement, or deterioration of circulation following an initial period of increased flow, should be evaluated by prompt angiography. Early reoperation is recommended to correct technical errors or to repair progressing disease.

REFERENCES

1. Barnes, R. W., G. D. Shanik, and Slaymaker, E. E.: An index in healing of below knee amputation: leg blood pressure by Doppler ultrasound. Surgery, *79*:13, 1976.
2. Beales, J. S. M., Adcock, F. A., Frawley, J. S., et al.: The radiological assessment of disease of the profunda femoris artery. Br. J. Radiol., *44*:854, 1971.
3. Berguer, R., Higgin, R. F., and Cotton, L. T.: Geometry, blood flow and reconstruction of the deep femoral artery. Am. J. Surg., *130*:68, 1975.
4. Bernhard, V. M., Militello, J. M., and Geringer, A. M.: Repair of the profunda femoris artery. Am. J. Surg., *127*:676, 1974.
5. Bernhard, V. M., Ray, L. I., and Militello, J. P.: The role of angioplasty of the profunda femoris artery in revascularization of the ischemic limb. Surg. Gynecol. Obstet., *142*:840, 1976.
6. Bernhard, V.M., Ray, L. I., and Towne, J. B.: The reoperation of choice for aortofemoral graft occlusion. Surgery, *82*:867, 1977.
7. Billig, D. M., Callow, A. D., and Deterling, R.: Surgical consideration in the management of lesions of the profunda femoris artery. Am. J. Surg., *119*:392, 1970.
8. Cotton, L. T., and Roberts, V. C.: Extended deep femoral angioplasty: an alternative to femoropopliteal bypass. Br. J. Surg., *62*:340, 1975.
9. Fernandes e Fernandes, J., Nicolaides, A. N., Angelides, N. A. et al.: An objective assessment of common femoral endarterectomy and profundaplasty in patients with superficial femoral occlusion. Surgery, *83*:313, 1978.
10. Graziano, J. L., Olander, G. A., and Lal, R. B.: Significance of profunda femoris artery in extremities with marked ischemia. Am. Surg., *35*:229, 1969.
11. Leeds, F. H., and Gilfillan, R. S.: Revascularization of the ischemic limb: importance of the profunda femoris artery. Surgery, *82*:25, 1961.
12. Lindbom, A.: Arteriosclerosis and arterial thrombosis in the lower limb: a roentgenological study. Acta Radiologica (Stockholm) Suppl., *80*:55, 1950.
13. Malone, J. M., Goldstone, J., and Moore, W. S.: Autogenous profundaplasty: the key to long-term patency in secondary repair of aortofemoral graft occlusion. Ann. Surg., *188*:817, 1978.
14. Martin, P. and Jamieson, C.: The rationale for and measurement after profundaplasty. SURGICAL CLINICS OF NORTH AM., *54*:95, 1974.
15. Martin, P., Frawley, J. E., Barabas, A. P., et al.: On the surgery of atherosclerosis of the profunda femoris artery. Surgery, *71*:182, 1972.
16. Martin, P., Renwick, S., and Stephenson, C.: On the surgery of the profunda femoris artery. Br. J. Surg., *55*:539, 1968.
17. Moore, W. S., and Hall, A. D.: Unrecognized aortoiliac stenosis. Arch. Surg., *103*:633, 1971.
18. Morris, G. D., Jr., Edwards, W., Cooley, D. A. et al.: Surgical importance of profunda femoris artery: analysis of 102 cases with combined aortoiliac and femoropopliteal occlusive diseases treated by revascularization of deep femoral artery. Arch. Surg., *82*:52, 1961.

19. Morris-Jones, W., and Jones, C. D. P.: Profundaplasty in the treatment of femoropopliteal occlusion. Am. J. Surg., *127*:680, 1974.
20. Roon, A. J., Moore, W. S., and Goldstone, J.: Below knee amputation: a modern approach. Am. J. Surg., *134*:153, 1977.
21. Strandness, D. E., Jr.: Functional results after revascularization of the profunda femoris artery. Am. J. Surg., *119*:240, 1970.
22. Thompson, B. W., Read, R. C., Campbell, G. S., et al.: The role of profundaplasty in revascularization of the lower extremity. Am. J. Surg., *132*:710, 1976.
23. Waibel, P. P., and Wolff, G.: The collateral circulation in occlusions of the femoral artery: an experimental study. Surgery, *60*:912, 1966.

The Medical College of Wisconsin
8700 West Wisconsin Avenue
Milwaukee, Wisconsin 53226

Management of Peripheral Arterial Aneurysms

Luis A. Queral, M.D., William R. Flinn, M.D.,†*
James S. T. Yao, M.D., Ph.D.,‡
and John J. Bergan, M.D.§

"The frequency and seriousness of its complications demand that a patent (peripheral) aneurysm be operated upon when diagnosed . . ."

PETER MARTIN[7]

The history of peripheral vascular surgery is highlighted by unique episodes in which man's perception of peripheral aneurysms has changed. For example, Galen described peripheral aneurysms in 170 A.D. and emphasized their predilection for disaster as he described, ". . . blood that spurted forth with much violence."[9, 35] Later, Antyllus first employed definitive surgical treatment by ligating the artery above and below the lesion. He then incised the aneurysm, evacuated the clot, and exteriorized the cavity.[21] This method of care was lost but the principles of care were revived in the sixteenth century by Ambrose Paré and then modified by Anel in 1710 and Philagrius, who first described actual excision of the aneurysmal sac.[9] Compression as definitive treatment was tried in the eighteenth and nineteenth centuries. This failed, of course, but the most extreme application was probably that of Parker,[23] who had teams of medical students apply pressure to the aneurysm continuously over a period of days. Even wise surgeons such as Bilroth in the 1860s and Halsted in the early twentieth century also employed forms of compression therapy. During the 1950s, peripheral aneurysms were excised and in the 1960s, they

*Fellow in Peripheral Vascular Surgery, Northwestern University Medical School, Chicago, Illinois
†Fellow in Peripheral Vascular Surgery, Northwestern University Medical School, Chicago, Illinois
‡Associate Professor of Surgery and Director, Blood Flow Laboratory, Northwestern University Medical School, Chicago, Illinois
§Magerstadt Professor of Surgery and Chief, Division of Vascular Surgery, Northwestern University Medical School, Chicago, Illinois

Supported in part by the Dr. Scholl Foundation and the Northwestern University Vascular Research Fund.

began to be bypassed as direct arterial reconstructive surgery matured.

Today, peripheral vascular surgery has progressed to the point where management of conventional arteriosclerotic femoral and popliteal aneurysms fails to excite much enthusiasm. However, in reviewing the subject from the vantage of this volume, one finds that a complacent attitude toward such aneurysms is not justified. In fact, two things have happened that have markedly altered the approach of the vascular surgeon to aneurysmal disease of the femoropopliteal system. The first is a change in cause and incidence of these aneurysms. The second is the rapid establishment of noninvasive imaging in confirmation of diagnosis.

This presentation will describe new ways in which society in general and the medical profession in particular have effected the therapeutic approach to aneurysms in the femoropopliteal system. Simplicity of diagnosis and surgical treatment now characterize management of these lesions.

TRUE ANEURYSMS OF THE FEMORAL ARTERY

Table 1 displays a modern classification of femoral artery aneurysms as observed at this time. Fusiform aneurysms of the common femoral artery are largely asymptomatic and are found incidental to routine physical examination. Although such lesions are relatively uncommon, it was possible to collect 63 such aneurysms in 45 patients at the Massachusetts General Hospital,[15] and 89 patients with femoral artery aneurysms were found at the Mayo Clinic through 1963.[31] These large series of patients give some indication as to the mode of presentation of patients with femoral aneurysms.

In the Massachusetts General Hospital series, 29 per cent of the lesions were entirely asymptomatic. Swelling, tenderness, and local pain were present in another 29 per cent. Rupture of the aneurysm was found in 14 per cent of the Massachusetts General Hospital cases but was only seen in five of the 95 aneurysms in the Mayo Clinic series.

More common than rupture of peripheral aneurysms is thrombosis. Aneurysms which cross joint creases cause occlusion of the artery that harbors them. In Tolstedt's group of 12 femoral aneurysms, eight thrombosed causing thigh amputation.[40] Pappas stated in his series

Table 1. *Femoral Artery Aneurysms*

Arteriosclerotic fusiform aneurysms of the common femoral artery and its bifurcation
Iatrogenic femoral artery aneurysms
False aneurysm following direct arterial puncture
False aneurysm following graft artery anastomosis to the femoral artery
Arteriosclerotic true aneurysms of the femoral artery after graft anastomosis to the femoral artery
Septic aneurysms of the narcotic addict

from the Mayo Clinic that there was a 16 per cent incidence of amputation related to complications of femoral aneurysms in a group of 44 patients who were considered unsuitable for surgery.[31]

Multiple aneurysms occur frequently in patients with femoral aneurysms. Crawford et al.[13] and Pappas in reporting from the Mayo Clinic both cited a 70 per cent incidence of multiple aneurysm while Dent, et al.[18] found that 95 per cent of their patients had multiple aneurysms. Cutler and Darling[15] found that half of their patients had aneurysms of the distal abdominal aorta and Pappas reported that 28 per cent of his 89 patients had abdominal aortic aneurysms. In the eight patients reported by Bergan, Kaupp and Trippel,[4] a total of 33 arteriosclerotic aneurysms was found; five of the eight patients had abdominal aortic aneurysms.

SURGERY OF TRUE ANEURYSMS

Surgical treatment of the arteriosclerotic fusiform aneurysm of the common femoral artery is based upon arteriographic findings. Multiple major branches are common. Doubled profunda femoris arteries or major circumflex iliac branches are not unusual. The principle of therapy is bipartite. First, the aneurysm must be excluded from the arterial circulation. Second, total restoration of arterial circulation must be accomplished.

Figure 1 indicates the variety of ways in which the distal arterial tree can be restored. When possible, the common bifurcation of the

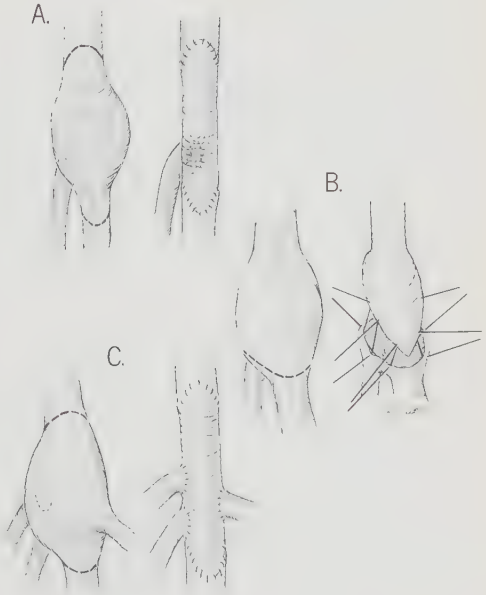


Figure 1. These diagrams illustrate the principle that all major outflow should be preserved as arteriosclerotic femoral aneurysms are resected. *A*, A discontinuous femoral artery bifurcation is shown. *B*, A common origin of the superficial femoral and profunda arteries is illustrated. *C*, Multiple major outflow branches have been encountered and carefully preserved.

superficial and deep femoral arteries should be incorporated so that the graft is a tube or sleeve. Expanded polytetrafluoroethylene (PTFE) or Dacron should be used. Vein grafts have been applied in this situation but they are too small to match accurately the size of the common femoral artery. Replacement of atherosclerotic aneurysms of the femoral artery with a Dacron or PTFE prosthesis is preferred. However, the exact reconstruction will depend on the anatomy and patency of the superficial femoral and profunda femoris arteries. The external iliac artery may be transected for an easy proximal end-to-end anastomosis. If an associated abdominal aortic aneurysm is present, femoral aneurysms may be replaced most effectively by aorto-femoral graft limbs.

IATROGENIC FEMORAL ANEURYSMS

At present, true atherosclerotic aneurysms of the common femoral artery and its branches are less common than are iatrogenic, mycotic, or postreconstructive pseudoaneurysms. This expansion of the etiologic horizon of aneurysmal disease of the femoral artery has resulted in an altered diagnostic and therapeutic approach to the patient with a pulsatile groin mass. Despite etiologic diversity, the pathophysiology of these lesions is straightforward. Persistent continuity between the arterial lumen and the surrounding soft tissue space is caused by transmural arterial trauma. Whether this trauma is direct or indirect, violent or controlled, the result to the patient is the same: a false aneurysm.

Puncture Aneurysms

In recent years, as the scope of invasive diagnostic and therapeutic procedures has broadened, iatrogenic false aneurysms have become very common. The percutaneous catheterization technique of Seldinger, modified for specific diagnostic and therapeutic maneuvers, frequently requires the vascular surgeon to evaluate the patient with a "hematoma" after such an arterial manipulation.[39] These lesions are most frequently seen in the groin, reflecting medical preference rather than anatomic predilection. Iatrogenic false aneurysms also occur in the upper arm or antecubital fossa when these anatomic sites are used for arterial access.

An obvious pulsatile mass at the puncture site may not be apparent, since extravasation of blood to a greater or lesser extent occurs after any arterial cannulation. Thus, clinical diagnosis of aneurysm may be difficult. Further, the severity of associated medical problems directly related to the indications for invasive study may delay the diagnosis initially.

If an aneurysm forms, neuropathic pain may be a striking initial symptom. Pain with distal radiation or paresis of distal muscle groups is infrequently encountered with true atherosclerotic aneurysms. However, such findings are common with iatrogenic false aneurysms. The

anatomic proximity of major nerves to arteries within the extremity allows an expanding mass lesion to cause early neurologic symptoms. The rapid rate of expansion of iatrogenic false aneurysms may account for development of this symptom. Its rarity in association with true aneurysms may be related to their slow growth.

Gray scale ultrasonography is remarkably useful in clarification of diagnosis in patients suspected of having arterial puncture aneurysms. Since it is not painful, is not invasive, and does not repeat an arteriographic event, it is acceptable to patients.

The diagnostic accuracy of gray scale ultrasonography is impressive. Continuity between the arterial lumen and surrounding anechoic areas confirms a diagnosis of aneurysm (Fig. 2) and excludes the diagnosis of hematoma (Fig. 3). Angiography may then be used with selectivity.

Usually, the lesion is simply a persistent, patent arterial puncture. After confirmation of diagnosis by ultrasound, the patient can be taken to the operating room. Proximal arterial control may be as simple as direct digital compression. Then, the aneurysm is evacuated and the small defect repaired by direct suture (Fig. 4). The problem and its solution have been so simplified that anxiety for the patient can be minimized.

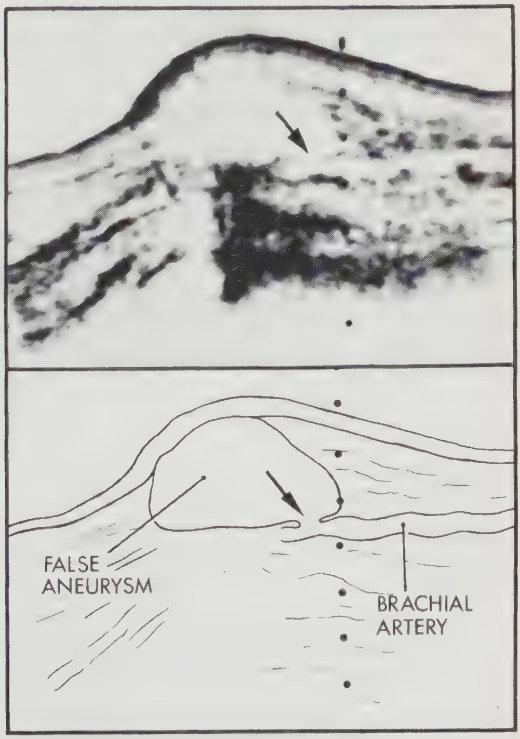


Figure 2. Gray scale sonography allows precision in the diagnosis of suspect puncture aneurysms. Here, there is clear continuity between the anechoic mass and the brachial arterial lumen. The distance between the dots is 1 cm.

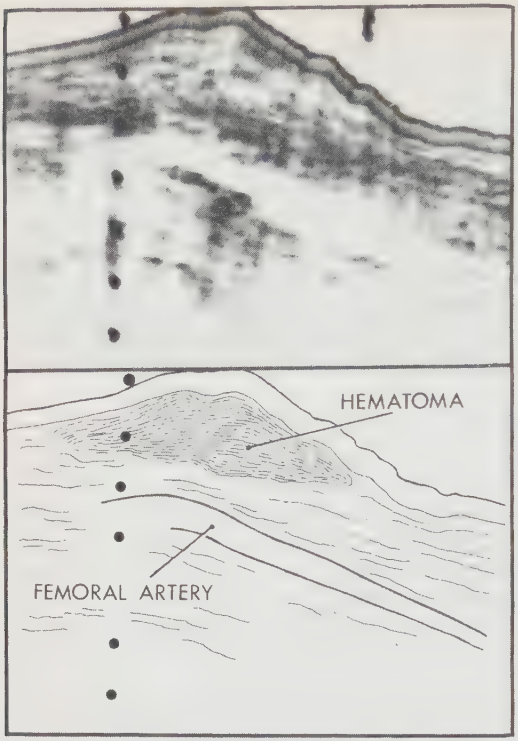


Figure 3. In the patient whose ultrasound scan of the femoral artery is shown here, the echogenic pulsatile mass is clearly not continuous with the underlying lumen of the femoral artery.

Anastomotic Iatrogenic Aneurysms

Arterial reconstructive surgery has expanded, technological advances have been made, but anastomotic false aneurysms remain. The common femoral artery is the most usual location for such postreconstructive false aneurysms.[8, 25, 34] Why this is true is not entirely clear. The differential compliance of synthetic prostheses as used in aorto-femoral and femoral-popliteal grafting may be one cause. Shear forces generated by end-to-side anastomosis may contribute.[38] Add to these the fact that the groin harbors the inguinal-femoral lymphatics which drain distal infected ischemic tissue and it is obvious that the groin is at highest risk for development of sepsis and subsequent anastomotic dehiscence (Fig. 5, *A* and *B*).

While various suture materials, techniques of implantation and grafts themselves have been indicated as causal in the formation of anastomotic aneurysm,[16] the single most important factor is the arteriosclerotic artery itself. The actual condition of the arterial wall and the progression of atherosclerosis may allow anastomotic failure.

Anastomotic aneurysms are infrequently associated with infection of grafts,[8, 25, 34] but, if infection of the suture line is present, dehiscence of the suture line and formation of a false aneurysm are a certainty.

Exposure of the aorto-femoral graft limb distal to the inguinal ligament, isolation of the subjacent common femoral artery, and skeleton-

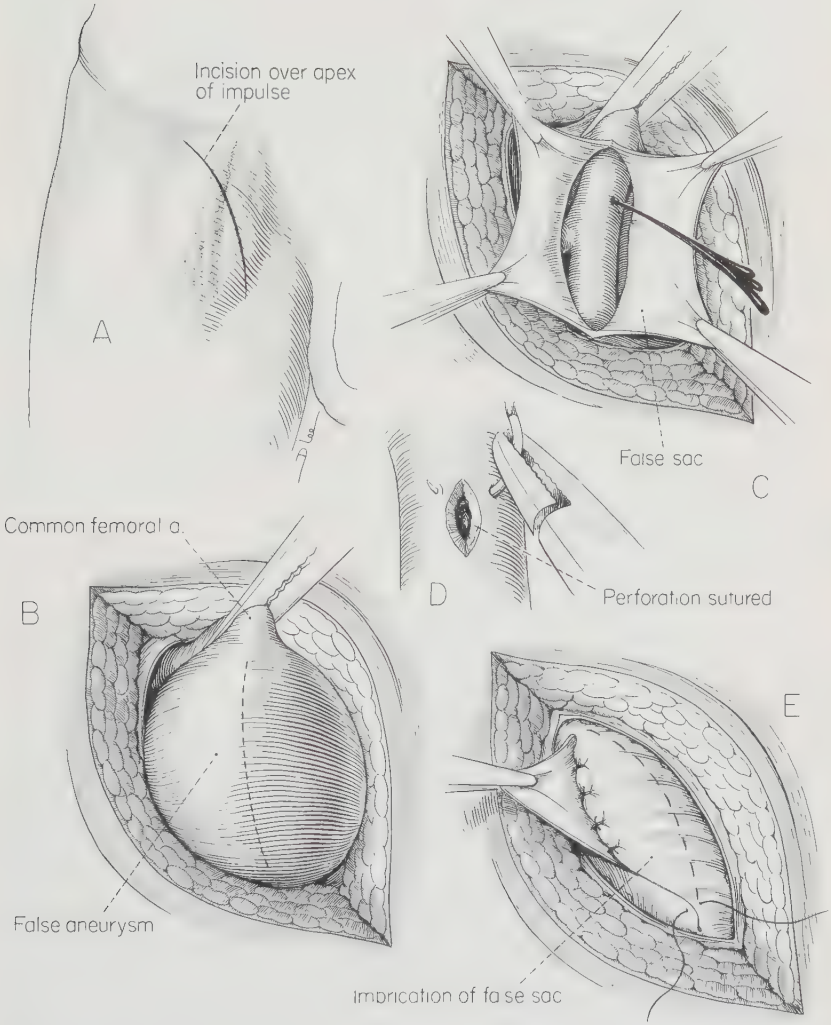


Figure 4. Surgical treatment of iatrogenic false aneurysms. Proximal control may be by clamp or even direct digital pressure. Simple suture closure of the arterial defect will be the only treatment necessary in most cases. (*From* Nora, P. F. (ed.): Operative Surgery. Philadelphia, Lea and Febiger, 1972, with permission.)

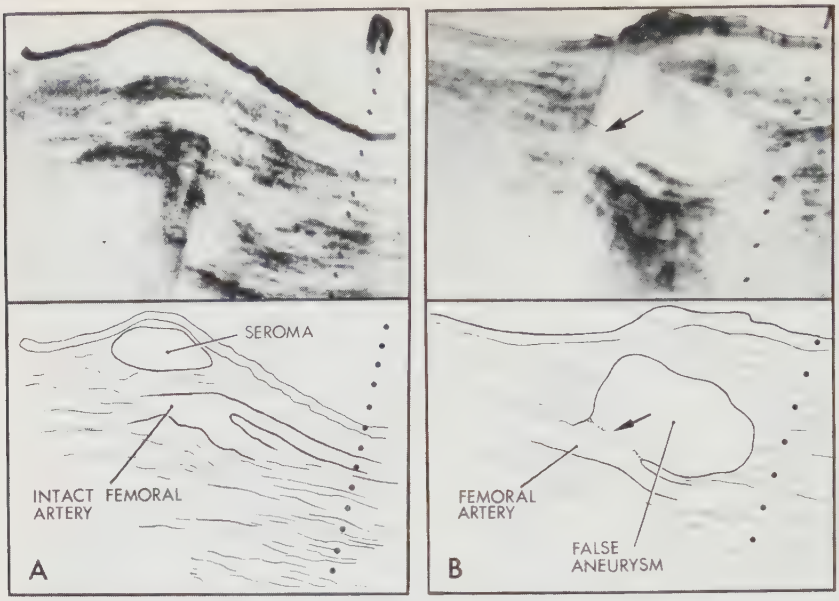


Figure 5. Ultrasound scanning can be of assistance in differentiating postoperative serum collections from actual anastomotic disruptions. *A*, The clear anechoic mass is well separated from the subjacent artery. *B*, Definite continuity between the arterial lumen and the aneurysm is demonstrated.

ization of the femoral branches in dissection of the anastomotic false aneurysm are not only tedious but unnecessary. In addition, separation of the previous anastomosis from the now adherent femoral vein is difficult. Therefore, simplification of the operation is desirable. To do this, one need only isolate the grafted limb and then control the common femoral artery and its branches by intraluminal balloon catheters. The grafted limb may be encircled distal to the inguinal ligament, proximal to the aneurysm. Alternatively, if the aneurysm is quite large, a proximal, lower abdominal curvilinear incision cutting the external oblique and internal oblique muscular fibers will allow exposure of the grafted limb in its retroperitoneal location.

Once the grafted limb is isolated and clamped, the aneurysm may be incised widely and the arterial orifices cannulated by the catheters (Fig. 6). Balloon tamponade is achieved and the balloons kept inflated by closure of three-way stopcocks.

In many cases, the deep femoral artery is the sole supply to the limb. It may be connected end-to-end to the graft. Frequently, an additional length of graft is required to complete the reconstruction.

TRUE ANEURYSM AFTER AORTO-FEMORAL BYPASS

One situation not usually anticipated in patients with prosthetic vascular grafts in the groin is now being observed as patients are followed for longer periods of time. This is a condition in which the anas-

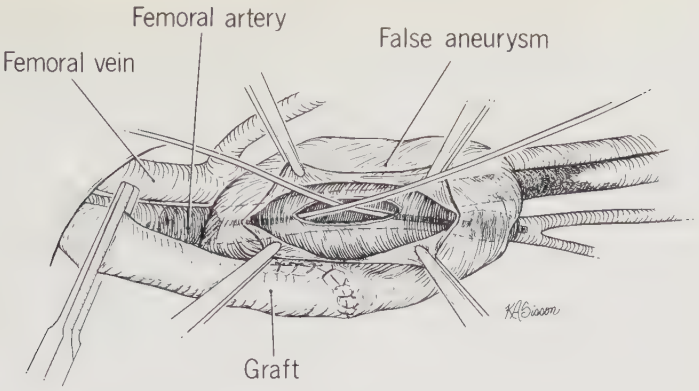


Figure 6. An anastomotic false aneurysm as viewed by the surgeon. Proximal common femoral artery occlusion and distal profunda femoris arterial control have been gained by balloon catheters. Note the graft occlusion by clamp and the commonly encountered superficial femoral artery occlusion.

tomotic suture line is intact and a true aneurysm of the femoral artery is found at or beyond the anastomosis. Such a condition might be anticipated in a patient who has had previous aorto-bifemoral grafting in treatment of abdominal aortic aneurysm. This coincides with our understanding of the natural history of aneurysmal disease, and the surgical treatment itself is governed by classical guidelines of peripheral aneurysm repair.

FEMORAL ANEURYSMS IN THE DRUG ADDICT

Widespread abuse of drugs in our society in recent times has resulted in production of some fascinating and challenging arterial lesions. In fact, infected pseudoaneurysms caused by parenteral drug abuse have become the most common type of mycotic aneurysm.[1] Of these, the commonest site is the femoral artery.[46] As peripheral veins become sclerosed, the addict seeks intravenous access through larger veins such as the femoral vein. Multiple injections into the same area become common because of inability to rotate injection sites. This increases the risk of septic vascular injury.

The formation of mycotic aneurysms may be the result of multiple unintentional arterial punctures. Aseptic technique is unknown to such patients and injection of agents that cause tissue necrosis compound the problem. Also, the arterial wall may be destroyed by contiguous spread of adjacent subcutaneous abscesses or areas of cellulitis produced by the septic injections.

Typically, these lesions appear as swollen, tender, erythematous areas overlying or near the course of a major artery. Pus and blood running from the groin should suggest the diagnosis of mycotic aneurysm of the drug addict. Leukocytosis may be profound (20,000 to 30,000). Examination of the feet may reveal the punctate purpuric le-

sions typical of septic embolization. These may in fact precede any local or systemic symptom of the mycotic aneurysm.

Recognition of the lesion at the time of presentation is critical to optimal treatment. Simple incision and drainage may be catastrophic. When bloody groin pus is seen, the physician should know that this is not just another subcutaneous abscess in a drug addict.

Angiographic confirmation of the diagnosis should follow administration of broad spectrum antibiotics. Prompt surgical treatment allows blood and wound cultures to be taken at surgery. Specific antibiotic coverage may begin later, when the organisms are identified. The causative microorganisms are extremely variable and reflect the gross contamination of the unsophisticated injection apparatus. Klebsiella, E. coli, Aerobacter, S. aureus, and S. epidermitis are seen. Anaerobes with unfamiliar nomenclature are common.

Infection may spread in a periarterial fashion along the limb. It is not unusual to find gas shadows along the course of the artery on plain x-ray examination. This demonstration of extensive spread of infection along tissue planes dictates surgical care.

Long ago, Paré established what has become the first principle in management of arterial infection. He not only used ligatures in treating contaminated vascular battlefield injuries but, when infection followed, he treated the patient with drainage and excision of the infected vessel. This principle should be followed in treatment of the arterial infection of the narcotic addict. Excision of infected arterial wall, wide debridement of local soft tissue and open drainage of the wound is done. No attempt is made at the initial operation to reconstruct distal arterial flow. The patient is monitored closely postoperatively by physical examination and Doppler pressure testing. While immediately postligation, ankle pressure will be zero, this will improve in a short time. Later, within 24 to 72 hours, an ankle pressure of 40 to 50 mm of mercury or greater will appear, and this is sufficient to maintain viability of the limb through the recovery period. Maintenance of collateral microcirculation is aided by the use of heparin and low molecular weight Dextran (10 ml per kg per 24 hr) in the intraoperative and postoperative periods.

Late arterial reconstruction is a problem in these patients. Previously infected tissue planes should be avoided and the graft must be hidden from the patient. If the graft is accessible in any way, the patient will soon use it for narcotic vascular access. Reconstruction by way of an obturator bypass as described by Fromm and Lucas has been successful in these patients.[20] It should be assumed that the patient will always be a drug addict.

INFECTED TRAUMATIC PSEUDOANEURYSMS

Penetrating injuries due to knife or gunshot wounds are now a common cause of infected traumatic pseudoaneurysms in cities. Infection may occur because of inadequate initial debridement, subsequent

myonecrosis, or use of prosthetic grafts.[29] The location of these lesions corresponds roughly to the location of arterial injuries as reported by Rich,[33] in which 56.8 per cent involved the lower extremities. Since all traumatic injuries near a major artery have a potential for causing a mycotic aneurysm, principles of surgical management must include early exploration after angiography, primary repair of arterial defects and avoidance of prosthetic materials. Aggressive suspicion of diagnosis of arterial injury will reduce significantly the incidence of secondary mycotic aneurysm.

PRIMARY MYCOTIC ANEURYSM

Rarely, a small group of mycotic aneurysms, the sequelae of primary arterial infection, may occur. Recognition of these aneurysms and their etiology was clarified by Sir William Osler, who coined the term "mycotic aneurysm" in presenting the first Gulstonian Lecture to the Royal College of Physicians in London, March, 1885.[30] He described the pathophysiology and correctly attributed the cause to septic emboli from infected cardiac valves. Such septic emboli may lodge in the vasa vasorum of peripheral arteries or on atherosclerotic intimal plaques. There, they may produce a gradual weakening of the arterial wall so that the formation of an aneurysm follows. Also, septicemia may infect already existing atherosclerotic aneurysms, with further destruction of the arterial wall and local contamination.[3] Surgical management, of course, follows the principles outlined above. The infected artery should be excised. Revascularization should be done through clean tissue planes if it is required.

POPLITEAL ANEURYSMS

Atherosclerotic aneurysms of the popliteal artery remain the most commonly encountered peripheral true aneurysms.[9] The clinical presentation, natural history, and guidelines for management have been thoroughly presented in the past.[15, 23, 41, 42] While a pulsatile mass may be found, the patient usually seeks the attention of a physician because of secondary complications. Ischemia of the limb, not always limb-threatening, due to thrombosis of the aneurysm is a common presentation. Also, localized distal ischemia with characteristic punctate purpura or localized digital gangrenous changes characteristic of atheroembolic phenomena should lead to a thorough search for a proximal aneurysm. Other indirect manifestations may include popliteal or femoral-popliteal vein thrombosis or neuropathic pain. Rupture remains an infrequent early symptom.

These lesions occur almost exclusively in men in their fifth and sixth decades. A high index of clinical suspicion coupled with a thorough physical examination may lead to early diagnosis of the uncomplicated lesion. The high incidence of bilaterality, 50 per cent or more,

and the extreme likelihood of proximal aorto-iliac aneurysmal disease in these patients must be kept in mind.

The use of ultrasonography in the diagnosis of these lesions has been well appreciated recently.[17, 36, 37] This should make the initial diagnostic approach clear. It is a simple matter for the ultrasonographer to scan the contralateral limb and the abdominal aorta at the time of initial screening. This gives valuable diagnostic information and allows structuring of the therapeutic surgical approach.

Angiography is essential to successful reconstructive efforts in patients with popliteal aneurysms. It may show extensive proximal sacculation and will define distal occlusion of tibial vessels. If untreated, these aneurysms develop complications in 30 to 75 per cent of cases.[14, 22, 45] Between 16 and 44 per cent[15] of these complications may result in amputation of the limb. Thus, surgical treatment must be strongly recommended if definitive diagnostic evaluation confirms presence of a popliteal aneurysm and if associated medical conditions do not preclude operative therapy.

In the past, popliteal aneurysms have been treated by resection and interposition of autogenous vein graft or Dacron graft. But it should be stressed that aneurysms need not be resected from the arterial circulation to effect a cure. It is simpler and equally effective to bypass the aneurysm with a standard femoral-popliteal below-knee reconstruction. The aneurysm is then trapped by proximal and distal ligation of the artery (Fig. 7).

Difficulties may arise in reconnecting distal popliteal or tibial vessels in patients with thrombosed aneurysms. Thromboembolic debris should be removed from these vessels and distal popliteal reconstruction performed. However, if this is unsatisfactory, distal tibial bypass to midcalf level can be performed with expectation of success. This

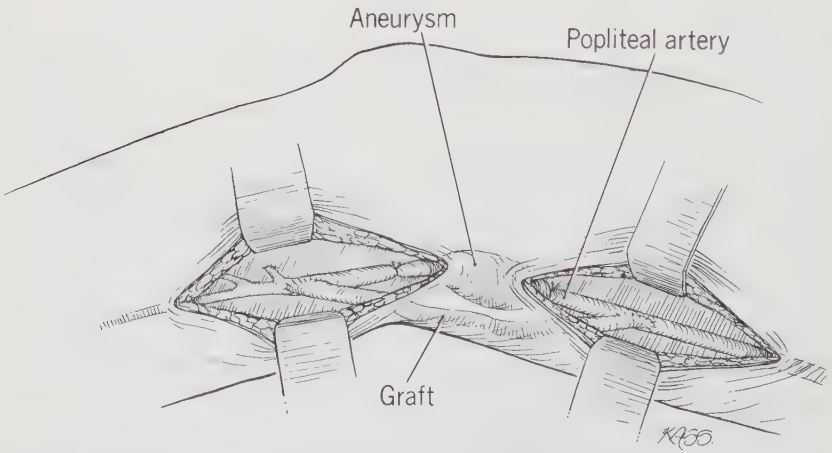


Figure 7. In the surgical treatment of popliteal artery aneurysms, the standard bypass is performed from the normal artery above the aneurysm to that below. The aneurysm is totally excluded from arterial circulation by proximal and distal ligature.

stresses the importance of delayed angiographic views to the ankle to prepare adequately for such a procedure. Intraoperative angiography is essential to ensure suitability of the distal circulation.

REFERENCES

1. Anderson, C. B., Butcher, H. R., and Ballinger, W. F.: Mycotic aneurysm. Arch. Surg., *109*:712, 1974.
2. Baue, A. E.: Obturator bypass. *In* Rutherford, R. B. (ed.): Vascular Surgery. Philadelphia, W. B. Saunders Co., 1977.
3. Bennett, D. E., and Cherry, J. K.: Bacterial infection of aortic aneurysm: A clinicopathologic study. Am. J. Surg., *113*:321, 1967.
4. Bergan, J. J., Kaupp, H. A., and Trippel, O. H.: Femoral aneurysmectomy. Management of the profunda femoris artery. Angiology, *20*:249, 1969.
5. Bernheim, B. M.: The ideal operation for aneurysm of the extremity. Report of a case. Bull. Johns Hopkins Hosp., *27*:93, 1916.
6. Bird, C. E.: Sympathectomy as a preliminary to the obliteration of popliteal aneurysm. Surg. Gynecol. Obstet., *60*:926, 1935.
7. Bouhoutsos, J., and Martin, P.: Popliteal aneurysm: A review of 116 cases. Br. J. Surg., *61*:469, 1974.
8. Chavez, C. M.: False aneurysm of the femoral artery. Ann. Surg., *183*:694, 1976.
9. Chitwood, W. R., Stocks, L. H., and Wolfe, W. G.: Popliteal artery aneurysm: Past and present. Arch. Surg., *113*:1078, 1978.
10. Crawford, E. S., DeBakey, M. E., and Cooley, D. A.: Clinical use of a synthetic arterial substitute in 317 patients. Arch. Surg., *76*:261, 1968.
11. Crawford, E. S., and DeBakey, M. E.: Popliteal arteriosclerotic aneurysm. Circulation, *32*:515, 1965.
12. Crawford, E. S., DeBakey, M. E., and Cooley, D. A.: Surgical considerations of peripheral arterial aneurysms. Arch. Surg., *78*:226, 1959.
13. Crawford, E. S., Edwards, W. H., DeBakey, M. E., et al.: Peripheral arteriosclerotic aneurysm. Am. Geriatr. Soc., *9*:1, 1961.
14. Crichlow, R. W., and Roberts, B.: Treatment of popliteal aneurysms by restoration of continuity. Ann. Surg., *163*:417, 1966.
15. Cutler, B. S., and Darling, R. C.: Surgical management of arteriosclerotic femoral aneurysms. Surgery, *74*:764, 1973.
16. Cutler, E. C., and Dunphy, J. E.: The use of silk in infected wounds. N. Engl. J. Med., *224*:101, 1941.
17. Davis, R. P., Neiman, H. L., Yao, J. S. T., et al.: Ultrasound scan in diagnosis of peripheral aneurysms. Arch. Surg., *112*:55, 1977.
18. Dent, T. L., Lindenauer, S. M., Ernst, C. B., et al.: Multiple arteriosclerotic arterial aneurysms. Arch. Surg., *105*:338, 1972.
19. Edmunds, L. H., Darling, R. C., and Linton, R. R.: Surgical management of popliteal aneurysm. Circulation, *32*:517, 1965.
20. Fromm, S., and Lucas, C.: Obturator bypass for mycotic aneurysm in the drug addict. Arch. Surg., *100*:82, 1970.
21. Garrison, F. H.: History of Medicine. Philadelphia, W. B. Saunders Co., 1929.
22. Gifford, R. W., Jr., Hines, E. A., Jr., and Janes, J. M.: An analysis and follow-up study of 100 popliteal aneurysms. Surgery, *33*:284, 1953.
23. Hardy, J. D., Tompkins, W. C., Hatten, L. E., et al.: Aneurysms of the popliteal artery. Surg. Gynecol. Obstet., *140*:401, 1975.
24. Keen, W. W., and White, J. W.: An American Textbook of Surgery. Philadelphia, W. B. Saunders Co., 1895.
25. Knox, W. G.: Peripheral vascular anastomotic aneurysm: A 15 year experience. Ann. Surg., *183*:120, 1976.
26. Lexer, E.: Die ideale operation des arteriellen und des arteriellvenosen aneurysma. Arch. Klin. Chir., *83*:459, 1907.
27. Linton, R.: The atherosclerotic popliteal aneurysm: Report of 14 patients treated by preliminary lumbar sympathectomy and aneurysmectomy. Surgery, *26*:41, 1949.
28. Matas, R.: An operation for radical cure of aneurysm based upon arteriorrhaphy. Trans. South. Surg. Assoc., *20*:396, 1902.
29. Mobius, G.: Specimen showing the effects of gunshot injury on the heart and blood vessels. Br. J. Surg., *8*:107, 1920.
30. Osler, W.: The Gulstonian Lecture on malignant endocarditis. Br. Med. J., *1*:467, 1885.

31. Pappas, G., Janes, J. M., Bernatz, P. E., et al.: Femoral aneurysms. Review of surgical management. J.A.M.A., *190*:489, 1964.
32. Pringle, J. H.: Two cases of vein grafting for the maintenance of a direct arterial circulation. Lancet, *1*:1795, 1913.
33. Rich, N., Baugh, J., and Hughes, C.: Acute arterial injuries in Viet Nam. 1000 cases. J. Trauma, *10*:359, 1970.
34. Richardson, J. V., and McDowell, H. A.: Anastomotic aneurysms following arterial grafting: A 10 year experience. Ann. Surg., *184*:179, 1976.
35. Rutherford, R.: Vascular Surgery. Philadelphia, W. B. Saunders Co., 1978.
36. Scott, W. W., Jr., Scott, P. P., and Sanders, R. C.: B-scan ultrasound in the diagnosis of popliteal aneurysms. Surgery, *81*:436, 1977.
37. Silver, T. M., Washburn, R. L., Stanley, J. C., et al.: Gray scale ultrasound evaluation of popliteal artery aneurysms. Am. J. Roentgenol. Radium Ther. Nucl. Med., *129*:1003, 1977.
38. Smith, R. F., and Szilagyi, D. E.: Healing complications with plastic arterial implants. Arch. Surg., *82*:34, 1961.
39. Thadani, U., and Pratt, E.: Profunda femoris arteriovenous fistula after percutaneous arterial and venous catheterization. Br. Heart J., 33:803, 1971.
40. Tolstedt, G. E., Radhe, H. M., and Bell, J. W.: Late sequela of arteriosclerotic femoral aneurysms. Angiology, *12*:601, 1961.
41. Tompkins, W. C., Smith, A. D., Wren, H. B., et al.: The atherosclerotic popliteal aneurysm. Am. J. Surg., *134*:813, 1977.
42. Towne, J. B., Thompson, J. E., Patman, D. D., et al.: Progression of popliteal aneurysmal disease following femoral popliteal aneurysm resection with graft. A 20 year experience. Surgery, *80*:426, 1976.
43. Wilson, S. E., Van Wegenen, P., and Passaro, E., Jr.: Arterial infections. Current Prob. Surg., *15* (No. 9), 1978.
44. Woodruff, M. F. A.: The Transplantation of Tissues and Organs. Springfield, Ill., Charles C Thomas, Publisher, 1906.
45. Wychulis, A. R., Spittell, J. A., and Wallace, R. B.: Popliteal aneurysms. Surgery, *68*:942, 1970.
46. Yellin, A.: Ruptured mycotic aneurysm, a complication of parenteral drug abuse. Arch. Surg., *112*:981, 1977.

Division of Vascular Surgery of the Department of Surgery
Northwestern University Medical School
303 East Chicago Avenue
Chicago, Illinois 60611

The Use and Abuse of the Vascular Laboratory

*D. E. Strandness, Jr., M.D.**

In recent years there has been increasing emphasis on the development and utilization of noninvasive testing in nearly all areas of the cardiovascular system. There are clear, compelling reasons for such advances since from the patient's standpoint a safe, painless method of evaluating cardiovascular function is obviously preferable to the alternative procedures. The prospects are excellent that with improving technology, the techniques will get even better in terms of identifying not only the site of disease but its physiologic consequences as well. It is not surprising that the utilization of these new approaches should provoke not only controversy regarding their usefulness but also result in outright abuses of the techniques.

The role the vascular surgeon occupies with regard to the problems of vascular disease is both unique and changing. In the area of arteriosclerosis obliterans he or she has traditionally provided the primary diagnostic evaluation as well the definitive therapy, in contrast to cardiac surgery in which patients are screened and in most instances entirely evaluated by the cardiologist prior to operation. Until recently, the same pattern has obtained in cases of extracranial arterial disease, when the neurologist is largely responsible for the workup. In the case of venous thrombosis and pulmonary embolism, the surgeon has generally been consulted only when the question of operative intervention is considered because of the failure of medical treatment.

These traditional roles for the vascular surgeon have changed dramatically in large part because of the development of noninvasive testing procedures. How did this come about? Because surgeons with few exceptions have been in the forefront of the development of many of these methods, they are now interposing themselves at several critical points in the evaluation process, rather than serving primarily as consultants at the stage when the workup is complete. Two examples clearly illustrate this change. Many of the major new methods of testing for extracranial arterial disease have been developed by surgeons and bioengineers. Thus, in many centers surgeons are involved at an

*Professor of Surgery, University of Washington School of Medicine, Seattle, Washington

early point in the evaluation of the patient, and the referring neurologists are increasingly dependent upon the information provided by the noninvasive laboratory.

Likewise in the area of acute venous thrombosis nearly every major advance in noninvasive testing has had its genesis in a surgical laboratory. Surgeons have, in fact, played a major role in educating the medical community about the vagaries of clinical diagnosis and the methods available of providing useful, objective information on which to base therapy.

Not surprisingly, the divisions within the medical community with regard to each of the above areas are sharp and often extremely rigid. There are some physicians who never use noninvasive testing, considering it to be irrelevant, inaccurate, and wasteful of time for both physician and patient as well as being fiscally irresponsible. The opposite end of the spectrum takes a different approach insisting that most, if not all, patients should have the tests performed as a routine part of the workup. It is important to review the current status of this growing field and attempt to reconcile some of the obvious differences of opinion.

SCIENTIFIC INQUIRY, OBJECTIVITY, AND THE POCKETBOOK

The field of noninvasive testing has been plagued with a series of problems which are not unique to this area but do merit consideration. It is possible to find conflicting reports about the accuracy of practically every proposed method. The best results are usually those emanating from the laboratory that either developed the method or was the first to use it. As other groups attempt to reproduce the results, it is not uncommon for an entirely different viewpoint to be expressed.

This is probably best explained in terms of the method of scientific inquiry and the degree of objectivity employed. Every scientist realizes the difficulties involved in testing a hypothesis, particularly when there is a bias involved, as there invariably is in practically every scientific endeavor. This can only be avoided if the protocol is set up in such a way as to be certain that some independent review is included. For objectivity it is mandatory that the results of the noninvasive test be compared whenever possible to some accepted standard. Further, the interpretation of results must be done independently and not solely by the investigators who are involved in proving the accuracy of the noninvasive test. It is imperative that the independent review stand alone, unchanged by the investigator, who may be biased in favor of his or her method.

Financial reward appears to be an increasing cause of bias. This comes about in two ways. First, the investigators may have a financial stake in the method being developed. Second, laboratories are being set up which have as their primary function profit rather than service

to the patient at a minimal cost. Neither of these situations is acceptable. With regard to the first issue, it is my view that journals considering reports on new methods should require a statement from the investigators as to whether or not they have a financial investment in the company marketing the device. Although it may be possible to be objective when there is a profit motive involved, suspicion may be aroused by such a financial stake.

All hospital-based laboratories must be established with the goal of service at minimal cost that is sufficient to cover the expense of equipment, necessary technical personnel, and supplies. It may be necessary to demonstrate that such services are not only cost effective but are helpful in the management of patients and may in fact reduce the overall cost to the patient. In some areas such as acute venous thrombosis, this is relatively easy to prove, whereas in many others it is more difficult to establish with certainty.

Over the past several years, studies have emerged from more than one center establishing the validity of certain concepts which should serve as the basis for some of the tests currently utilized. Since these facts differ depending upon the area of the circulation involved, it is important to review them.

PERIPHERAL ARTERIAL DISEASE

Arterial Pressure

With the exception of arterial aneurysms, diseases of the arterial system result in the development of regional hypotension distal to the involved segment.[6] Furthermore, if the degree of involvement worsens, the pressure distal to the sites of involvement falls even further.[18] Conversely, if the collateral circulation improves or the areas of occlusion are bypassed, the distal pressure will increase. Thus, as in the case of hypertension, the best, simplest method of following the progress of clinically evident disease is by measuring the arterial pressure at the convenient site furthest removed from the disease, which may be at the ankle, forefoot, or digit.

The noninvasive methods most commonly used to measure limb pressure permit an estimate only of systolic pressure but this is not a serious drawback, since systolic pressure is the most sensitive indicator of change in the status of the disease. Systolic limb pressure can be measured with an ultrasonic velocity detector or a variety of simple plethysmographic systems. Further, it is feasible to make the determinations at all levels of the limb down to and including the digits.[12, 17, 25] The equipment necessary to make these measurements can be purchased for $500 or less depending upon individual preference and experience.

Is the measurement of limb blood pressure important to the vascular surgeon? If the determination of arm blood pressure is useful in following the course of hypertension (a fact difficult to dispute), it is

equally important for the surgeon to measure ankle blood pressure to follow the progress of regional hypotension. The reasons are obvious since regional hypotension reflects not only the degree of impairment but is the most sensitive and reliable indicator of change in the disease with or without operative therapy.[6] Further, there is increasing evidence that the absolute pressure levels may also have important therapeutic implications.

Arterial Flow

Arterial flow to the limb can be expressed in terms of total volume flow (cc per minute), regional, such as to the calf or digits (cc per 100 cc of tissue per minute), and velocity (peak, mean, and its forward-reverse flow relationships). For clinical purposes, the only method useful as a routine is to evaluate the velocity changes in the major arteries at all levels of the limb. This is usually done by examining the arteries at the sites usually palpated for either no flow or a monophasic signal which always indicates problems proximal to the site being examined. This may be done quickly at the same time the ankle pressures are being measured and provides the surgeon with useful evidence as to the level of the arterial occlusion and the patency of the run-off vessels.

The measurement of total limb flow or regional blood flow by plethysmography or isotope methods provides no additional useful information for the surgeon. It is true that such studies can be used to describe the physiologic consequences of arterial obstruction, but they are much too cumbersome for routine use.

Research is proceeding on quantification of arterial velocity patterns at various levels of the limb as a method of more clearly defining the level and extent of arterial involvement, particularly as related to the type of operative procedure to be employed.[9, 14] While the initial reports are encouraging, it is premature to advocate routine use of such techniques for such purpose until they have been more widely tested and proven useful.

Limb and Digit Volume Changes

Arterial disease modifies the response of the extremity to the flow increase induced by each myocardial contraction. The volume pulse waveform loses its sharp systolic peak and dicrotic wave.[18, 21] However, the absolute volume change shows considerable overlap with normal readings and thus adds little to simple examination of the waveform changes themselves.[21] Although there are systems that can provide such data for the digits, forefoot, and limbs, there is no convincing evidence that they add any additional information which is of more value than the measurement of systolic pressures, except that measurement of systolic pressure in the digits is useful in distinguishing primary from secondary Raynaud's phenomen.[6] Also, there is increasing evidence that pressures measured at the toe level may be useful in predicting whether or not an open lesion will heal.

Stress Testing

It is evident both in the coronary and peripheral arterial circulation that adding stress either by exercise[20] or a reactive hyperemia test[8] is the best method of assessing the functional capacity of the available collateral circulation. In the case of the legs, the response to such stress is best monitored by measurements of systolic pressure at the ankle.[19] Treadmill exercise provides the following additional information that may be helpful: the time of onset of claudication; maximal walking time; location of the pain and walking pattern; and baseline values for future comparison. It is also useful in patients with suspected neurogenic claudication in whom the clinical picture may at times be confusing.[11]

Whether one uses a reactive hyperemic test or treadmill exercise is a matter of personal preference.[8, 20] The drop in pressure that occurs with both is similar but the recovery time is not as rapid when exercise is used which is as expected. The treadmill test in combination with resting pressures is useful for following the progress of the collateral circulation and the results of direct arterial surgery. An important question that remains to be answered is whether the exercise test is more sensitive than resting pressures alone in detecting of the progression of disease.

EXTRACRANIAL ARTERIAL DISEASE

Since stroke is the second most common cause of death due to cardiovascular disease, it is important to make an accurate anatomic diagnosis in order to insure the institution of proper therapy. The important clinical categories include transient ischemic attacks, completed stroke, asymptomatic bruits, nonspecific cerebrovascular symptoms and signs, and vertebro-basilar insufficiency. Before considering the problems involved in noninvasive testing for these categories, it is important to clarify the current ideas with regard to the causes of cerebral ischemia. In brief, it can occur either with hemodynamically significant lesions or with those that do not reduce flow but may be the source of emboli. Thus, any technique that will detect only flow reducing lesions of greater than 50 per cent diameter reduction will miss a considerable number of plaques that may be responsible for transient ischemic attacks and strokes.

A key question is whether there is any justification for noninvasive testing if an arteriogram is to be performed regardless of the findings.[19] The answer at present is clearly no, unless the technique provides information that will be useful for follow-up purposes to evaluate either the progression of disease or the results of therapy. All of the indirect tests, including the supraorbital Doppler test,[5] oculoplethysmography-phonoangiography,[15] and oculopneumoplethysmography[10] suffer from the same serious limitations: they will detect only high grade stenoses or occlusions; they cannot distinguish a stenosis

from an occlusion; once positive, they remain so, and thus cannot be used for follow-up purposes to detect progression; and they do not provide information that would change the indications for arteriography.

It is important at this point to make a few comments about the reports of these tests and their accuracy. There is clear evidence of the limitations of the supraorbital Doppler test and oculopneumoplethysmography. Also, as Gee[10] has correctly concluded, oculopneumoplethysmography will be positive only with lesions sufficient to reduce pressure. More difficulty is encountered in the assessment of the accuracy of oculoplethysmography and carotid phonoangiography.

The combined system of evaluating ocular pulses and bruits is theoretically attractive since if the two complement one another it may be possible to separate internal carotid from external carotid disease as well as separate the high grade lesions of greater than 50 per cent diameter reduction from those which produce a bruit but do not result in a delay in the ocular pulse. Indeed, the developers report an overall accuracy of 90 per cent with this system.[15] We have used this method in a prospective study and found it completely unsatisfactory as a screening test for patients with suspected extracranial arterial disease. The test was positive in only 17 of 40 vessels (42 per cent) with a high grade stenosis (greater than 50 per cent diameter reduction).[4] Further, the addition of bruit recording at three levels was disappointing and did not add to the overall accuracy of the system. It was concluded from our studies in which the arteriograms were reviewed independently of the test that the method should not be used as either a screening test or for the evaluation of the symptomatic patient, and has been abandoned entirely in our institution.

What of the asymptomatic bruits discovered at the time of a routine physical examination or prior to a major operation? If the supraorbital Doppler test or oculopneumoplethysmography are unequivocally positive and if the bias of the surgeon is for an aggressive approach to patients in this category, arteriography is probably warranted.

The newer ultrasonic imaging methods hold much greater potential for the evaluation of patients with suspected carotid disease. These include pulsed and continuous wave flow imaging,[2] B-mode scanning,[16] and combined B-mode and pulsed Doppler ultrasonic systems.[3] A great deal more work remains to be done before final conclusions can be drawn but certain facts appear to be emerging: it is a relatively simple matter to distinguish a high grade stenosis from an occlusion; lesions that do not reduce pressure or flow may be detectable if they result in a disturbance in flow; it may be possible to follow patients sequentially to evaluate the progression of disease; and the tests will be suitable for evaluating the results of carotid endarterectomy in terms of restenosis. In spite of the promising data reported to date, it is premature to recommend widespread purchase and use of these systems except in those centers familiar with the technology and in which the capability exists for the conduction of careful prospective trials. These devices must still be considered experimental.

ACUTE VENOUS THROMBOSIS

With the introduction of the labeled fibrinogen test, there was an explosion of renewed interest in this field.[13] One of the great achievements in the last 10 to 15 years has been an accumulation of data that focuses attention on the inaccuracy of bedside diagnosis of acute venous thrombosis. While phlebography is the best single test, its use is limited by its hazards, a fact which has led to a search for a simple, reliable noninvasive test which could replace it. Considerable progress has been made and continues in defining the role of both noninvasive tests and those situations in which phlebography must be performed. The techniques that have been developed include Doppler ultrasound,[1] impedance[24] and strain gauge plethysmography,[23] and phleborheography.[7] Several excellent studies have shown that these tests can detect thrombi in the popliteal vein and higher in the body with a degree of accuracy sufficient to warrant the institution of anticoagulant therapy without requiring phlebography. Likewise, a normal examination, particularly with repeat testing, may be sufficient to rule out a diagnosis of major deep venous thrombosis.

An important question which must be raised is the problem of isolated thrombosis of the calf vein which cannot be detected without the use of labeled fibrinogen or phlebography. Since the fibrinogen test must be used prior to the event, it is of no value in the patient who presents with symptoms. Zielensky et al.[26] found the impedance method to be insensitive to thrombi in this area detecting only 18 per cent, whereas the sensitivity of the Doppler method was 55 per cent. Thus, although a positive test is reliable, a negative test in a symptomatic patient requires a phlebogram to make a correct diagnosis.

DISCUSSION

The application of noninvasive testing methods for evaluation of arteriosclerosis obliterans, extracranial arterial disease, and acute venous thrombosis must be considered in terms of the setting in which they are used. The surgeon who wishes to utilize such methods in an office setting can derive a considerable amount of useful information using a pocket-sized Doppler instrument with suitable cuffs for pressure measurements. This method can be used as an adjunct to the physical examination, requiring very little extra time. In addition, it is possible with experience to recognize the different types of velocity patterns that are observed on both the arterial and venous side of the circulation.

In the hospital, there is a legitimate need for a noninvasive laboratory that can serve as a consultative service to evaluate aspects of arterial and venous disease which are often a source of confusion and uncertainty. However, it must be recognized that such a laboratory requires knowledgeable personnel who are experienced in this discipline. The staff must have a broad knowledge of not only the diseases

being evaluated but the problems associated with the instrumentation required. The practice of buying the equipment, hiring a technician to do the tests, and hoping for the best is to be discouraged. The function of a good laboratory is to provide objective data for different medical specialists who encounter patients with suspected vascular problems but do not have the expertise to make a precise diagnosis.

Useful data which may be of some value to the surgeon include:

The level and extent of arterial occlusion. An ankle-arm pressure index of greater than 0.5 is seen most commonly with single level disease. Also, a quick survey of the velocity patterns at each level is useful. It only takes a few minutes to determine if those arteries of concern to the surgeon are patent or occluded.

Perfusion pressure to the distal limb. Absolute levels less than 50 mm of mercury should alert the surgeon that flow is at best marginal and that the patient should be watched very closely since pain at rest frequently develops and if an open lesion were to appear, healing might be a problem.

The potential for healing of a toe or foot ulcer. There is accumulating evidence that healing will occur with systolic pressures above levels of 55 mm of mercury at the ankle in nondiabetic patients. In patients with diabetes, the ankle pressure is not a good indicator of prognosis. However, toe pressures were noted to have greater predictive value as shown by Carter.[6] In his study, there was only one amputation in 21 patients with digit systolic pressure above 55 mm mercury. Conversely, it must be remembered that pressures below this level cannot be taken as certain indicators that healing will not occur. In those extremities with toe pressures below this level, approximately two-thirds went on to heal without amputation. Thus pressures at the ankle and toe are helpful but not absolutely predictive of the potential for healing.

The differentiation of neurogenic from true intermittent claudication.[11] The measurement of ankle pressures in patients whose symptoms are atypical may be useful in distinguishing those patients suffering from neurospinal disease. The finding of a normal ankle pressure before and after exercise rules out arterial disease as the cause for the leg complaints. Even in patients with combined disease, exercise testing may be helpful. For example, if the patient is stopped because of pain secondary to neurospinal disease, the decrease in pressure at the ankle is usually much less and the recovery time quicker than in patients with true claudication.

The immediate success of direct arterial surgery as assessed in the operating suite and the recovery room.[22] Early problems may be detected by monitoring flow in the graft itself and measuring the systolic pressure at the ankle. If the ankle-arm pressure index falls during the postoperative period or a velocity signal in a graft disappears, this is certain evidence of occlusion. However, in patients with multisegment disease in which only the most proximal disease is corrected, the systolic pressure at the ankle may not change appreciably in the first 12 to 24 hours.

Detection of the progression of disease or of the failure of a graft during follow-up.[20] A fall in the ankle-arm pressure index is certain evidence of new problems. All of the above information can be obtained with a velocity detector that costs less than $500, with tests requiring only a few minutes to perform.

With regard to extracranial arterial disease, the current systems can be relied upon to detect only high grade stenoses or occlusion. A positive test will, with a high degree of accuracy, predict the presence of a hemodynamically significant lesion but a negative test must be ignored. In the symptomatic patient, these tests will not alter the indications for arteriography. Further, since the tests remain positive, they are of little value in following the progress of the arterial disease. If one adopts the attitude that patients with bruits who are found to have high grade stenoses should be operated upon, the noninvasive tests can be useful in selecting these patients for arteriography. With the progress now being made with ultrasonic imaging systems, it is likely that the supraorbital Doppler and plethysmographic systems will soon become obsolete. Newer systems will be able to distinguish a stenosis from an occlusion and also detect those lesions that do not reduce pressure or flow. When this becomes a reality, it will be possible to recommend ultrasonic imaging for all patients with suspected extracranial arterial disease. It makes little sense to purchase systems that do not change the indications for arteriography and cannot be used for long-term follow-up.

On the venous side of the circulation, noninvasive testing methods appear to be well on their way to becoming an established approach to this critically important area. They provide a simple, safe, and quick assessment of the state of the major deep veins. The surgeon must, however, be certain that a test has been performed adequately and fully understand the significance of the findings. In the event of a questionable result, or in a clinical situation in which exact localization of the involvement is required, phlebography should be ordered immediately.

It cannot be emphasized too strongly that, as in all tests in medicine, there are confidence limits and also that errors will inevitably occur. Clinical judgment is as important here as in any other area of surgery. While there is little doubt that the results of well performed tests will in many circumstances aid the surgeon in making a therapeutic decision, the patient and the clinical presentation must not be forgotten. In ordering such tests, it is important for all of us to realize that these are added to the patient's bill.

The decision to open a vascular laboratory must be made based upon the local needs of the institution and on the availability of well trained personnel to run the laboratory and carry out the tests. Improperly performed tests are a waste of both money and resources and may lead to incorrect diagnoses and therapy. A rush to buy all the latest equipment is to be condemned; tests of equipment must be shown to be reproducible by investigators not associated with the development of the method.

Finally, with regard to the future, it is clear that the entire field

will continue to advance as technology improves. However, it is hoped that potential users will recognize that proper application requires a detailed understanding of the limitations, sensitivity, and specificity of the methods to be used.

REFERENCES

1. Barnes, R. W.: Doppler ultrasonic diagnosis of venous disease. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
2. Barnes, R. W., Bone, G. E., Reinertson, J. E., et al.: Noninvasive ultrasonic carotid arteriography: Prospective validation by contrast arteriography. Surgery, *80*:328, 1976.
3. Blackshear, W. M., Phillips, D. J., Thiele, B. L., et al.: Detection of carotid occlusive disease by ultrasonic imaging and pulsed doppler spectrum analysis. Surgery, in press.
4. Blackshear, W. M., Thiele, B. L., Harley, J. D., et al.: A prospective evaluation of oculoplethysmography and carotid phonoangiography. Surg. Gynecol. Obstet., *148*:201, 1979.
5. Brockenbrough, E. C.: Periorbital Doppler velocity evaluation of carotid obstruction. Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
6. Carter, S. A.: Role of pressure measurements in vascular disease. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
7. Cranley, J. J.: Diagnosis of deep venous thrombosis of the lower extremity by phleborheography. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
8. Fronek, A., Coel, M., and Bernstein, E. F.: Postocclusive reactive hyperemia and the toe pulse reappearance time in the evaluation of arterial disease. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
9. Fronek, A., Coel, M., and Bernstein, E. F.: The importance of combined multisegmental pressure and Doppler flow velocity studies in the diagnosis of peripheral arterial occlusive disease. Surgery, *84*:840, 1978.
10. Gee, W.: Clinical application of ocular pneumoplethysmography. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
11. Goodreau, J. J., Greasy, J. K., Flanigan, D. P., et al.: Rational approach to the differentiation of vascular and neurogenic claudication. Surgery, *84*:749, 1978.
12. Gundersen, J.: Segmental measurements of systolic pressure in the extremities including the thumb and the great toe. Acta. Chir. Scand. (Suppl.), *426*:1, 1972.
13. Hobbs, J. T., and Davies, J. W. L.: Detection of venous thrombosis with 131 I-labeled fibrinogen in the rabbit. Lancet, 2:134, 1960.
14. Johnson, K. W., Maruzzo, B. C., and Cobbold, R. S. C.: Errors and artifacts of Doppler Flowmeters and their solution. Arch. Surg. *112*:1335, 1977.
15. Kartchner, M. M., and McRae, L. D.: Clinical application of oculoplethysmography and carotid phonoangiography. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
16. Mercier, L. A., Greenleaf, J. F., Evans, T. T., et al.: High-resolution ultrasound arteriography. A comparison with carotid arteriography. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
17. Nielsen, P. E., Bell, G., and Lassen, N. A.: The measurement of digital systolic pressure by strain-gauge technique. Scand. J. Clin. Lab. Invest., *29*:371, 1972.
18. Raines, J. K., Darling, R. C., Buth, J., et al.: Vascular laboratory criteria for the management of peripheral vascular disease of the lower extremities. Surgery, *79*:21, 1976.
19. Sandok, B. A.: Noninvasive techniques for diagnosis of carotid artery disease (editorial). Stroke, *9*:427, 1978.
20. Strandness, D. E., Jr.: Exercise testing in the evaluation of patients undergoing direct arterial surgery. J. Cardiovasc. Surg., *11*:192, 1970.
21. Strandness, D. E., Jr., Waveform analysis in the diagnosis of arteriosclerosis obliterans. *In* Peripheral Arterial Disease: A Physiologic Approach. Boston, Little, Brown and Co., 1969.

22. Sumner, D. S.: Preoperative, intraoperative, and postoperative monitoring of patients with arterial disease. *In* Rutherford, R. B. (ed.): Vascular Surgery. Philadelphia, W. B. Saunders Co., 1977.
23. Sumner, D. S.: Volume plethysmography in vascular disease: An overview. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
24. Wheeler, H. B.: Impedance phlebography: The diagnosis of venous thrombosis by occlusive impedance plethysmography. *In* Bernstein, E. F. (ed.): Noninvasive diagnostic techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
25. Yao, J. S. T., and Takaki, H. S.: Techniques of measuring lower limb arterial pressures. *In* Bernstein, E. F. (ed.): Noninvasive Diagnostic Techniques in Vascular Disease. St. Louis, C. V. Mosby Co., 1978.
26. Zielinsky, A., Hull, R., Hirsh, J., et al.: Comparative study of doppler ultrasound (DP) and impedance plethysmography (IPG) in the diagnosis of symptomatic and asymptomatic deep vein thrombosis (DVT), Abstract 453, II-117. Circulation II, 58: no. 4, Oct. 1978.

Department of Surgery
University of Washington School of Medicine
Seattle, Washington 98195

Lumbar Sympathectomy

Role in the Treatment of Occlusive Arterial Disease in the Lower Extremities

*Anthony M. Imparato, M.D.**

In 1923, Royle and Hunter of Australia performed the first lumbar sympathetic denervation in a man to ameloriate unilateral spastic paralysis by division of the white ramus from the second lumbar nerve, the gray rami of the second, third, and fourth lumbar nerves, and of the sympathetic trunk below the fourth lumbar ganglion. Incidental vascular changes noted in the skin of the leg consisting of increased warmth, deepening of the normal pink color and the appearance of capillary dilatation, disappeared by the 17th day. Their report stimulated Adson and Brown to develop lumbar sympathectomy for patients with vasospastic disorders of the extremities. Their procedure was to include bilateral ramisectomies, excision of the sympathetic trunks including the second, third, and fourth ganglia, and the outer sheaths of the common iliac vessels on each side for distances of 5 cm. Clinical responses were striking. Thermocouples and calorimetry were used to estimate blood flow to the skin. Skin temperatures rose 2° to 7° centigrade while heat production and radiation increased 200 to 900 per cent. Improvement persisted for three to six weeks. By 1925, they had followed five improved patients for up to three years.[5]

Coincidentally, Diez of Buenos Aires, aware of the unsatisfactory attempts by Jaboulay and Leriche of Lyon to effect lower extremity sympathetic denervation by periarterial stripping, did sympathectomies in patients with thromboangiitis by removal of the second lumbar to the third sacral ganglia, empirically based on anatomical rather than physiologic evidence, and relieved two patients of unremitting severe pain.[5]

*Professor of Surgery, Department of Surgery, New York University Medical School; Attending Surgeon, New York University Hospital; Visiting Surgeon, Bellevue Hospital, New York, New York; Consulting Surgeon, Patterson General Hospital, Patterson, New Jersey, Norwalk Hospital, Norwalk, Connecticut, Veterans Administration Hospital, New York, New York

Thereafter, sympathectomy was done for all types of ischemic disease of the lower extremities[3, 4, 5] until the introduction of direct arterial intervention for arterial occlusions which resulted in such dramatic improvement that sympathectomy became relegated to a secondary role. Experience with arterial reconstructions and precise angiographic delineation of occluded arteries revealed a group of patients who were not suitable for arterial reconstruction, in whom immediate amputation was unacceptable for the relief of pain at rest, ischemic ulcers, and limited gangrene of toes and feet, who were candidates for sympathectomy.[1, 10, 18, 21] Sympathectomy to improve results of arterial reconstructions,[20] to lower the levels of amputation, and to treat claudication[11] were also done.

Results were difficult to evaluate since the procedures were performed in a heterogenous population by a variety of surgical techniques, at different stages of ischemia resulting from a variety of causes, with different criteria for success. Criteria ranged from relief of claudication to lowering the level of amputations, encompassing relief of pain at rest, healing of ischemic ulcers, and avoidance of amputation, leading to great confusion regarding the proper role of sympathectomy in ischemic occlusive arterial disease of the lower extremities.

The inherent physiologic complexities of the sympathetic nervous system, in which the response of the blood vessels is to a neurohumeral transmitter, noradrenaline, which is also produced by the adrenal medulla and circulated throughout the body, added to the complexities of the pathologic processes which lead to ischemia, intensify the problems of evaluating results. Indeed, blood vessels respond as well to histamine, acetylcholine, pituitary ergotoxin, pain, muscular activity, cold and hypoglycemia. Additionally, ablation of the sympathetic nerves is often followed shortly by the return of vasomotor tone probably through the reorganization of alternate sympathetic pathways.[13] There is the added problem of increased sensitivity of sympathetically denervated end organs to circulating norepinephrine.[13]

A body of empirically derived experience suggests that patients with severe chronic ischemia of the extremities who are not suitable for direct arterial reconstruction may benefit from lumbar sympathectomy to achieve healing of ischemic lesions, relief of ischemic pain at rest, avoidance or postponement of amputation, and perhaps even improvement in walking ability. Results are not universally achievable. Definitions of success and failure and elaboration of predictive criteria are needed.

ANATOMY

The sympathetic system, composed of afferent and efferent fibers, is a link in a series of reflex arcs (Fig. 1). Afferent (sensory) fibers come from blood vessels and certain integumental structures and are carried in somatic nerves to cell bodies in the dorsal root ganglia of the spinal nerves. Efferent fibers arise from cells in the anteromediolateral

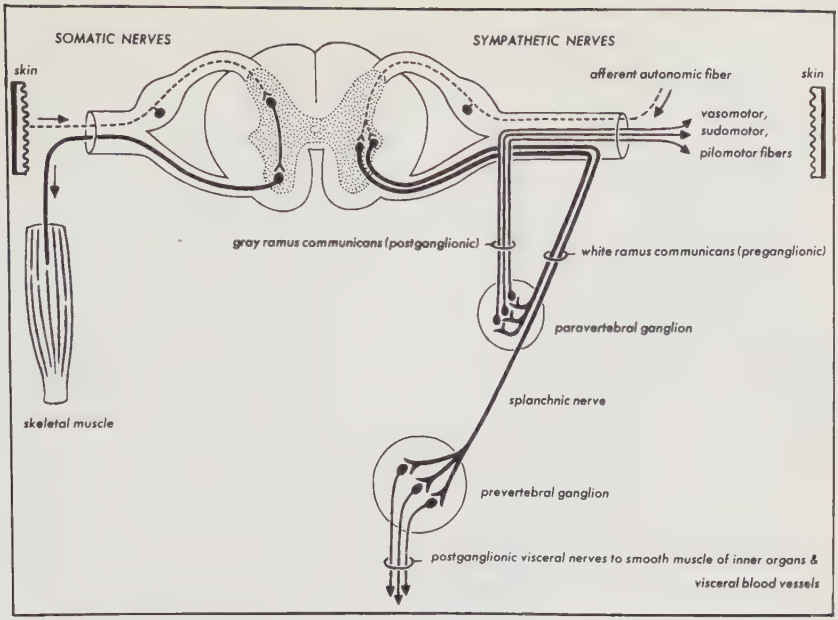


Figure 1. The general arrangement of afferent and efferent nerves in the sympathetic nervous system contrasts with the arrangement of somatic nerves. *From* Pick, Joseph: Identification of Sympathetic Segments. Ann. Surg., *145*:355, 1957, with permission.

columns of the spinal cord from the eighth cervical to the second or third lumbar segment to synapse with postganglionic fibers in the lumbar chain. Preganglionic innervation of the lower extremities is from D-10 to L-2 or L-3 and no other easily identified preganglionic fibers have been identified going to the chain (Fig. 2). Preganglionic axons are carried in the anterior nerve roots and synapse with neurons lying in the sympathetic ganglia. Three groups of ganglia, vertebral, prevertebral and terminal are connected to each other by nerve trunks and to the spinal nerves by rami communicantes. White rami communicantes carrying myelinated preganglionic fibers limited to the thoraco-lumbar outflow leave the spinal cord via anterior nerve roots. The gray rami leave the chain segmentally at each thoracolumbar and sacral level to S-3, carrying postganglionic unmyelinated fibers back to the appropriate spinal nerves for distribution segmentally according to dermatomes, to sweat glands, erector pili muscles, and blood vessels of skeletal muscle and skin. Prevertebral ganglia lie anterior to the aorta around its major branches and are the celiac, superior mesenteric, aortorenal and inferior mesenteric ganglia. Terminal ganglia lie near the urinary bladder and rectum. Ganglia called intermediate ganglia are in the thoracolumbar region outside the paravertebral ganglionated chain, variable in number and location, usually in close proximity to communicating rami, anterior spinal nerve roots and peripheral nerves and not readily accessible to surgical resection.[16] In the lumbar region they may be within the psoas muscle. They are susceptible to pharmacologic blockade.

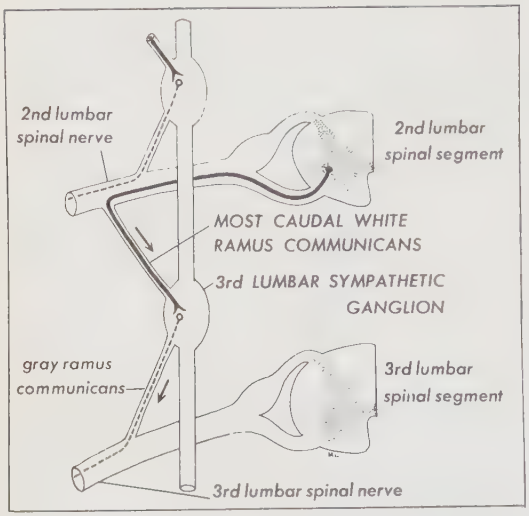

2nd lumbar spinal nerve
2nd lumbar spinal segment
MOST CAUDAL WHITE RAMUS COMMUNICANS
3rd LUMBAR SYMPATHETIC GANGLION
gray ramus communicans
3rd lumbar spinal segment
3rd lumbar spinal nerve

Figure 2. The arrangement of preganglionic and postganglionic fibers in the lumbar region illustrates the lowermost preganglionic fiber from L_2 in a Y configuration under the crus of the diaphragm. *From* Pick, Joseph: The Autonomic Nervous System. Philadelphia, J. B. Lippincott Co., 1972, with permission.

Pathways for integration of autonomic reflexes are at spinal, medullary, hypothalmic, and cortical levels. In the limbs, the primary function appears to be heat regulation through its effect on arterioles and sweat glands mediated through a system of arteriovenous fistulae located in the skin.

Diffuse discharge possible through the sympathetic system is explained by the manner of synapse in ganglia. Each preganglionic fiber synapses in only one ganglion at levels which may be far removed from its issuance from the spinal cord. For the lower extremity, the lowermost preganglionic fiber may be L-1, 2, or 3 while synapses with as many as 20 postganglionic cells occur along the entire length of the lumbosacral sympathetic chain and in intermediate ganglia. Postganglionic fibers issue segmentally from L-1 to S-3 to be distributed to peripheral end organs via peripheral nerves and along blood vessels. Synaptic innervation may also overlap in that one ganglion cell may be supplied by several preganglionic fibers. Sympathetic fibers surrounding the aorta derived from splanchnic nerves are difficult to trace, are in continuity with fibers surrounding major arteries that are derived from segmental postganglionic fibers and may represent another source of sympathetic fibers not accounted for by removal of the lumbar sympathetic chain alone (Fig. 3).

PHYSIOLOGY

In the extremities, the vascular response to sympathetic stimulation is vasoconstriction with blanching and cooling of skin and increased sweating, whereas blocking of the system results in increased blood

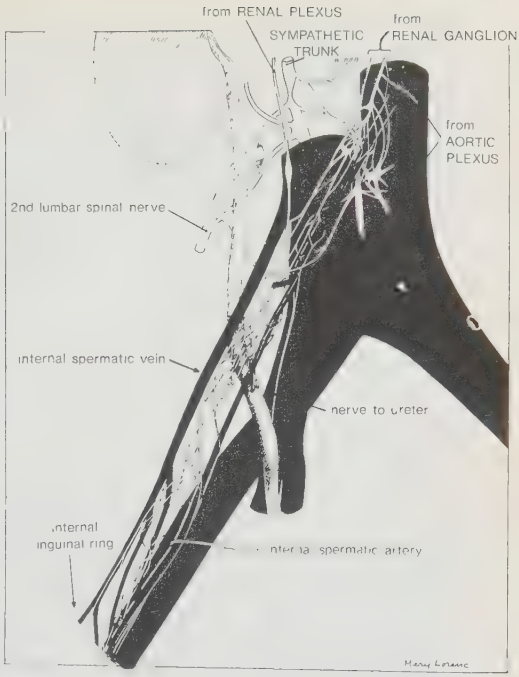


Figure 3. Sympathetic fibers from the ganglionated chain are intermingled with those from the periaortic plexus at the level of the iliac arteries. *From* Pick, Joseph: The Autonomic Nervous System. Philadelphia, J. B. Lippincott Co., 1972, with permission.

flow through cutaneous arteriovenous fistulae and cessation of sweating, thereby resulting in increased dryness, warmth and accentuation of pink color. Effects on blood flow to the muscles are considered incidental to the effect on flow in the skin and are overshadowed by the local effects of muscle metabolites. The effector organ can be influenced by circulating substances similar to the neurohumoral transmitter.

Adrenergic transmitters, classified as catecholamines, may be either excitatory or inhibitory, depending primarily upon whether they act upon so called alpha or beta receptor sites found in smooth muscle. Norepinephrine is primarily excitatory, isoproterenol primarily inhibitory, and epinephrine both excitatory and inhibitory. Control of skin is chiefly mediated through alpha receptors while blood vessels of the muscular bed are influenced through beta receptors. Alpha blockers should be expected to mimic sympathetic ablation.

Other neurohumoral transmitters are suspected to exist. Removal of the sympathetic system in experimental animals results in peripheral vessels regaining intrinsic vasomotor tone, and may make manifest an increased sensitivity to administered epinephrine. When the sympathoadrenal system discharges, blood is shifted from the skin and splanchnic bed to skeletal muscle, actions produced as well by epinephrine secreted by the adrenal medulla. In man, the effects of local lumbar sympathetic denervation vary greatly in extent and duration, evidenced by early clinical reports which described the vasomotor effects of sympathectomy disappearing as early as 17 days in patients

Figure 4. Acceleration of nail growth occurred following a successful lumbar sympathectomy performed for inoperable occlusive arterial disease.

with spastic paralysis and persisting for as long as six weeks in those with vasospastic disorders. In the presence of occlusive disease, the variability is even greater with patients several years later after lumbar sympathectomy for occlusive arterial disease manifesting warmer, drier, and pinker skin on the side that has been operated upon.

Whether sympathectomy increases nutritive blood flow to the skin is debated since increased flow is through arteriovenous fistulae. Nevertheless, an unreported observation here included shows acceleration of toenail growth following successful lumbar sympathectomy in a patient with inoperable occlusive arterial disease (Fig. 4).

PATHOLOGIC PROCESSES

Arterial lesions resulting in ischemia of lower extremities are varied, progressive at different rates in different individuals, and may be complicated by neural lesions as in diabetes mellitus. Whether the neural degeneration involves the autonomic nervous system as well, resulting in autosympathectomy, is debated. Buerger disease is subject to arrest by cessation of smoking. Mild ischemia from whatever cause manifested by intermittent claudication responds favorably to walking

and cessation of smoking. The course of ischemic symptoms varies greatly with the specific pathologic process, such that patients with thrombosed popliteal aneurysms may have a good prognosis because of relative noninvolvement of tibial arteries while patients with diabetes who tend to have occlusions of tibial arteries constitute the majority of patients who come to amputation. Patients with diabetes suffer an added limb threatening susceptibility to necrotizing infections, even in the absence of major arterial occlusions.

Such is the variability of the pathologic processes for which lumbar sympathectomies are performed. It illustrates the pitfalls of applying physiologic data directly to pathologic clinical conditions.

SELECTION OF PATIENTS

Intermittent claudication does not signify the advent of progressive ischemia. Indeed, patients with claudication will improve or remain stable and relatively rarely require amputation. They therefore are not generally considered for surgical intervention. Patients with advanced ischemia resulting in pain at rest, ischemic ulcers, frank gangrene, or impending gangrene are surgical candidates. Since arterial reconstruction provides dramatic relief, angiographic studies to assess its feasibility are indicated. Those suitable are so treated while those with contraindications because of angiographic patterns are considered for sympathectomy. In the face of threatened loss of a limb, indications for arterial surgery are extended to include utilization of blind popliteal segments,[12] single tibial arteries, and malleolar vessels.[9] When runoff in such patients has seemed questionably adequate, lumbar sympathectomy has been performed first, and if not effective then arterial reconstruction has been done.

In spite of the autosympathectomy question, diabetes mellitus does not appear to be a contraindication to sympathectomy. Persistent vasomotor tone can be demonstrated in patients with diabetic neuropathy.[7] The presence of diabetic spreading necrotizing infection is a contraindication until its control by debridement. Thereafter if revascularization is not possible and localized gangrene or ulceration persist, sympathectomy is considered.

LUMBAR SYMPATHECTOMY — PREDICTIVE TESTS

Predictions of the outcome of lumbar sympathectomy can be classified as those which define the type and distribution of the pathologic process, those which assess the degree of vasomotor tone, and those which estimate the extent of collateral circulation.

Assessment of Degree of Ischemia and Extent of Pathologic Process

Although there is some disagreement regarding whether the degree of ischemia is useful in predicting the outcome of lumbar sym-

pathectomy, it is generally agreed that short of extensive gangrene proximal to the forefoot, regardless of pulse patterns or walking ability, any patient with pain at rest, ischemic ulcers, or localized forefoot gangrene may respond favorably to lumbar sympathectomy.

In selecting diabetic patients with pain at rest and in evaluating surgical results, it is important to differentiate ischemic pain from the pain of diabetic neuropathy by determining the effect of gravity upon the symptoms. Neuropathic pain is not influenced by either the recumbent position or by hanging the foot over the side of the bed while ischemic pain is influenced favorably by having the foot in the dependent position. Relief of ischemic pain at rest, healing of ulcers regardless of how long it takes, and the avoidance of major amputation compared to suitable controls should be used as end points.

Angiographic patterns have no clearcut predictive value regarding the outcome of lumbar sympathectomy although patients with localized occlusions of the femoral area fare best. In these patients, however, the question is raised of whether surgical intervention was required at all. On the other hand, patients considered inoperable for arterial reconstruction because of extensive occlusion of tibial and pedal arteries have improved following lumbar sympathectomy.

Tests of Vasomotor Tone

Assessment of vasomotor tone by the responsiveness of skin temperature, electrical conductivity of skin, sweating, plethysmographically determined skin blood flow or vascular resistance in response to a number of maneuvers has been tried with varying success for many years. Various nerve blocks including posterior tibial block, lumbar paravertebral block, and epidural and spinal anesthesia have at best given approximately 70 per cent correlation. Responses to changes in environmental temperature are time consuming to determine and have sometimes yielded unsatisfactory correlations. Responsiveness to pharmacologic agents, most recently to an alpha blocker (Moxislyte hydrochloride (Opilon) administered intravenously was reported to have only a 40 per cent correlation, in which often a poor test response was followed by a good response to sympathectomy.[26] Marked decrease in pedal vascular resistance (PARI)[18] following sympathectomy suggests an additional predictive test which though requiring a complex technique with complex equipment not now generally available, merits additional investigation.[6, 8, 14, 18, 25]

Assessment of Collateral Circulation

ANGIOGRAPHY. Rough estimates of the collateral circulation around major arterial blocks can be made from angiographic study. These are difficult to quantitate accurately and are of no major value in predicting a response to sympathectomy.

DOPPLER ULTRASOUND TESTS. Determination of ankle pressures and ankle brachial pressure ratios is a useful technique easily performed. Ankle systolic pressure is measured by the Doppler ultrasound method. The Doppler flow probe is used as a stethoscope over the pos-

terior tibial and dorsalis pedis arteries. Audible signals are obtained even when pedal pulses are absent by palpation. A blood pressure cuff is then applied around the ankle above the malleoli and inflated to a pressure above that of the brachial artery. Ankle systolic pressure is determined at the level at which flow signals return during a subsequent deflation of the cuff. Pressure index is calculated by dividing the ankle systolic pressure by the brachial systolic pressure. Yao and Bergan found that the ankle pressure index was unrelated to the extent of tissue necrosis or to the nature of the initial complaint. Patients with gangrene, pregangrene or cutaneous ulcers had pressure indices of more than 0.25 while patients with pain at rest, and no necrosis might have indices of less than 0.20. Seventy-eight per cent of those who had ankle pressure indices of greater than 0.25 had favorable responses to sympathectomy. There also was no correlation of the results of sympathectomy with the presence or absence of gangrene or ulceration while there was a close correlation with ankle pressure index. Those who came to amputation in spite of sympathectomy had ankle pressure indices of less than 0.20, while those with indices above 0.35 universally had good responses. They conclude, however, that although their determinations did not identify all patients who had a good response to sympathectomy, it identified patients who are probably hopeless candidates, namely those whose ankle pressures are zero.[26] This group should probably not be subjected to sympathectomy under any circumstances.

The effect of sympathectomy on proliferation of collateral circulation is debated on the basis of clinical and experimental observations. If there is no collateral circulation of note at the time of sympathectomy, the procedure will be ineffective. The most useful and easily obtainable predeterminants of the efficacy of sympathectomy include the extent of gangrene involving the foot and estimates of the collateral circulation by measurements of ankle pressures. Gangrene extending proximal to the forefoot is an absolute contraindication to sympathectomy, since even were improvement to occur, an effective walking surface of the foot could not be salvaged. Absolute ankle pressure measurements and determinations of ankle brachial indices are indicated in all prospective candidates for sympathectomy. Those with zero ankle pressures should not be operated upon. Those with pressure indices of 0.20 or less have only a 30 per cent chance of improving, whereas those with 0.35 or greater can be expected to have excellent responses to sympathectomy. Those with pressures between 0.20 and 0.35 in whom salvage is predictable in a smaller percentage of cases should be evaluated in terms of general condition and ability to tolerate a possibly ineffective procedure.

SURGERY

The marked variability of the lumbar sympathetic ganglia (Fig. 5) and the presence of intermediate ganglia outside the main ganglionated lumbar chain determined the extent of lumbar sympathectomy

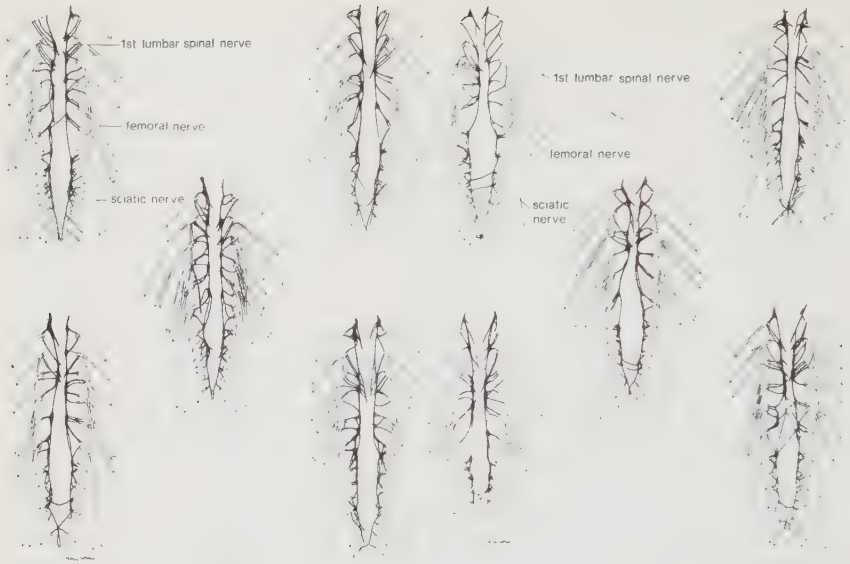


Figure 5. Morphology of the lumbar sympathetic chain varies markedly. *From* Pick, Joseph: The Autonomic Nervous System. Philadelphia, J. B. Lippincott Co., 1972, with permission.

which we have performed, and was first suggested to us by Dr. Joseph Pick in 1960.[15, 16] It is, in essence, the operative procedure first performed by Royle and Hunter, consisting of excision of the preganglionic fiber found under the crus of the diaphragm together with the paravertebral chain from the level of the crus to the crossing of the iliac vessels, achieving a postganglionic sympathectomy for much of L-2 to L-4 and preganglionic sympathectomy for L-5 to S-3 since that portion of chain is not excised. Since the intermediate ganglia are not recognizable at operation, it is hoped that removal of the lowermost preganglionic fiber will interrupt preganglionic connections of the lumbar intermediate ganglia affecting a preganglionic sympathectomy for whatever intermediate ganglia there might be. The chain is excised to include removal of cross communicating fibers between the sympathetic trunks.[24]

With the patient in the semilateral position, care is taken not to compress the lateral femoral cutaneous nerve or peroneal nerves. Pillows are placed between the knees, and the kidney rest is elevated to permit distraction of the iliac crest from the costal margin. A muscle-splitting incision is made from the end of the eleventh rib to the border of the ipsilateral rectus sheath at the level of the umbilicus. Each muscle layer is widely undermined, the transversalis fascia is incised, and the peritoneal contents are mobilized anteriorly to the level of the diaphragm, while the kidney is mobilized to an anterior location. Care is taken to come anterior to the psoas muscle. The ureter is retracted anteriorly. The lumbar sympathetic chain is identified at approximately the level of L-3 by palpation and exposed from the level of the crossing of the iliac vessels to the crus of the diaphragm. Fibers attached to

the chain are divided. The crus is divided inferiorly, exposing a Y-shaped configuration of the chain comprising the ganglionated main trunk coming vertically downward and the preganglionic fiber entering the lateral aspect of the chain obliquely from above. Both fibers are transected, liberating the chain for its removal. Frozen section study of the removed tissue is immediately performed (Fig. 6).

The operative procedure is quickly performed, in an attempt to keep total operating time to 30 to 45 minutes to avoid prolonged stasis in compromised arteries in the extremities.

Complications which can be anticipated include lacerations of the lumbar and iliac veins and the inferior vena cava on the right side. Lumbar veins especially under the crus of the diaphragm may cross anterior to the sympathetic chain and should be divided between metal hemostatic clips. Injuries to the inferior vena cava or iliac veins should be controlled by digital or sponge compression of the cava above and below the site of injury and carefully sutured with arterial suture material. Injuries to the ureter should be repaired at once under the direction of a urologist.

On the left side, emboli may be dislodged from the aorta and iliac vessels. Removal of the lumbar chain of lymph nodes rather than the sympathetic chain may be avoided by noting the semitranslucent appearance of the lymphatic channels, by the pattern of lymphatic vessels, which differs somewhat from that of the sympathetic nerves, and by pathologic examination in frozen section of the excised chain.

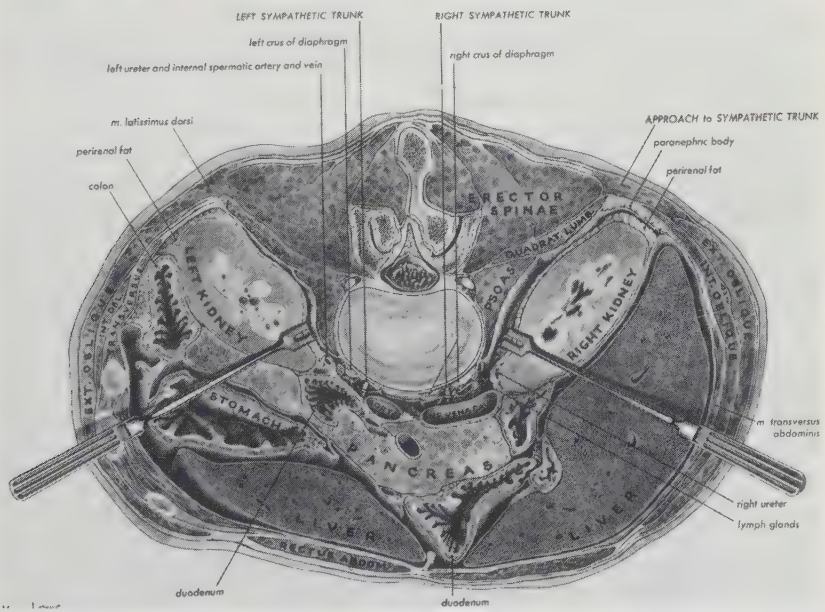


Figure 6. A surgical approach to the lumbar sympathetic chain necessitates anterior mobilization of the kidney, taking care not to dissect behind the psoas muscle. The relationship of the crus of the diaphragm, major vessels, and ureters is shown. *From* Pick, Joseph: The Autonomic Nervous System. Philadelphia, J. B. Lippincott Co., 1972, with permission.

Severe vertebral osteoarthritis with marked lipping may deform and split the chain, requiring careful dissection for its complete removal. Retroperitoneal hemorrhage may occur especially in patients on anticoagulant therapy so that special care must be used to achieve absolute hemostasis. Paradoxical gangrene has never been observed by the author. In one patient in whom lumbar sympathectomy was unduly prolonged, ischemia became markedly worse, precipitating surgical intervention in a situation previously thought to be inoperable. The resulting arterial reconstruction had an immediate but short-lived success, failing after approximately three weeks and resulting in amputation. Paradoxical gangrene has variously been ascribed to progression of arterial occlusion, to redistribution of blood between the limbs, termed hemometakinesia,[3] and to short circuiting of blood through precapillary arteriovenous shunts, thereby decreasing effective nutrient flow.

Postsympathectomy pain usually begins one to two weeks after sympathectomy, is often nocturnal and generally remits spontaneously within three months. It is deep and boring in nature involving the thigh and may require narcotics, though diphenylhydantion (Dilantin) and carbamazephine (Tegretol) have been used with improvement.[17]

Bilateral removal of the lowermost preganglionic fibers may result in failure of ejaculation, an unpredictable complication to be considered in younger males.

Mortality from lumbar sympathectomy ranges from one to 6.5 per cent, is usually from cardiac or pulmonary complications and occurs usually in the very aged.

Postoperative abdominal distention is usually a result of paralytic ileus and may require nasogastric intubation for two to three days, but any accidental entry into the peritoneal cavity may raise the suspicion of mechanical intestinal obstruction.

Local care of ischemic areas of the feet is carefully continued during the preoperative and postoperative periods, protecting the heels of the feet to prevent decubiti which may precipitate major amputations.

Chemically achieved sympathectomy through injections around the lumbar chain, most recently reported by Walker et al.[22] who used 6.7 per cent phenol in water, warrants serious consideration to supplant surgical sympathectomy. The site of injection is selected with the aid of biplane image intensification fluoroscopy. Injections can be repeated if necessary. In spite of the close proximity of vital structures, no evidence of damage has been detected except occasionally to neighboring somatic nerves resulting in transient neuropathy.

RESULTS

The results of lumbar sympathectomy are difficult to evaluate and comparisons amongst reported series are equally difficult. Those performed for inoperable arterial occlusions are the easiest since they are easily identified, have had arteriographic classification and are in ter-

minal phases of illness when outcome is more readily apparent. There is agreement that half to two-thirds of these patients who are subjected to lumbar sympathectomy, whether or not they have diabetes, will have relief of pain at rest, healing of ischemic ulcers, and improved ability to walk.[8, 10, 18, 19, 21] More controversial is the question of whether amputation can be avoided. Kim[10] et al. reported a 40 per cent amputation rate upon failure of sympathectomy, suggesting by implication that amputation might be avoided by sympathectomy. Berardi[1] et al. reported that sympathectomy is unlikely to delay amputation, which occurred invariably in their patients with diabetes. Szilagyi agreed that the amputation rate was not significantly higher in patients with diabetes after sympathectomy than in patients who did not suffer from the disease.

It is generally agreed that the site of ultimate amputation cannot be changed from above to below the knee by sympathectomy. In the absence of frank gangrene at the below-knee site, successful amputation can be performed in nearly 90 per cent of patients regardless of pulse patterns without sympathectomy. In the light of this, series reporting beneficial effects of sympathectomy are suspect in that other factors may be involved besides sympathectomy. Whether transmetatarsal amputations are made possible by sympathectomy is not clearly established.

Most controversial is the question of improvement of intermittent claudication by sympathectomy.[11] The natural course of intermittent claudication is one of improvement or stability so that without carefully controlled series of nonsurgically treated claudicators, it is difficult to accept improvement ascribed to sympathectomy. In addition, physiologic experiments fail to show increased blood flow to the muscles following sympathetic blockade.[20, 25]

The ability of ankle pressure measurements to predict relief of pain at rest and healing of ulcers is approximately 70 per cent, representing a modest improvement over the results of other published series, but suffers from the fact that 30 per cent of patients who have unfavorable ratios respond to sympathectomy and 15 per cent with favorable ratios do not. The greatest value of these determinations is in eliminating from consideration for operation those patients whose ankle pressures are zero who universally can be expected to come to amputation in spite of sympathectomy.

A criticism of ankle pressure measurements is that patients with diabetes have calcified tibial arteries making their accurate compression by cuff difficult and causing pressure readings to be falsely high. Nevertheless, it is recommended that ankle brachial pressures be taken and ratios calculated in patients undergoing lumbar sympathectomy.

Explanations proposed to account for immediate failures of sympathectomy include incomplete operation related to the anatomical factors previously described, autosympathectomy occurring in patients with diabetes although their vasomotor tone can be shown to persist,[7] and absence of sufficient collateral vessels to respond to denervation.

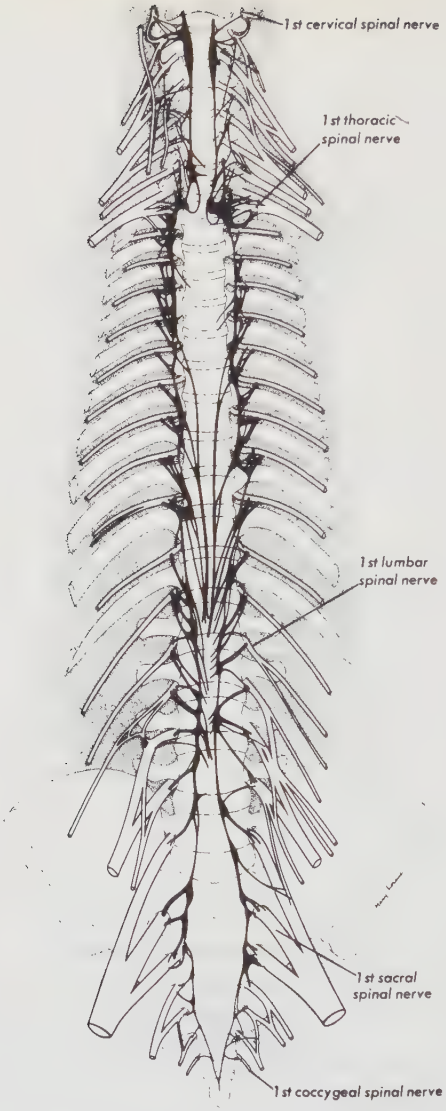

1st cervical spinal nerve
1st thoracic spinal nerve
1st lumbar spinal nerve
1st sacral spinal nerve
1st coccygeal spinal nerve

Figure 7. The entire ganglionated paravertebral sympathetic chain extending into the pelvis illustrates the nature of sympathetic ablation produced by operations currently in vogue: postganglionic upper and midlumbar segments, preganglionic distal lumbar and sacral segments. *From* Pick, Joseph, and Sheehan, D.: Sympathetic Rami in Man. J. Anat., *80*:12, 1946, with permission.

Marked variations in surgical techniques of sympathectomy make comparisons amongst series difficult. Standardization of the operative procedure would seem desirable, and the one proposed appears to answer all objections except that of failure to perform periarterial stripping, since postganglionic sympathectomy is achieved for the thigh and leg while preganglionic sympathectomy results for the critical foot

dermatomes L-4-S-2 (Fig. 7). At variance with physiologic data is the observation that in spite of the foregoing, patients in whom sympathectomy fails often show warmth to the level of the malleoli while the feet remain cold.

Hypersensitivity to circulating sympathetic substances has been proposed to explain late failures. Although hypersensitivity is a major factor in experimental animals, it is generally conceded that it is not a major factor in humans. The progressive nature of the process of arterial occlusion with worsening of ischemia complicates the interpretation of results. Nevertheless, it is clear that the most striking effects of sympathectomy are noticeable in the early postoperative period. Some patients, however, show striking differences in color and temperature between extremities many years after sympathectomy. No explanation is readily forthcoming. Incomplete sympathectomy must still be considered. Preganglionic sympathectomy alone does not seem to alter results. The progressive nature of the arterial disease, and trauma to ischemic limbs which may result in progressive gangrene complicate the evaluation of long term results. For this reason, sympathectomy should not be considered a definitive procedure but rather ancillary to a program of total care with careful attention to the general condition of the patient, to local care of the foot, to the avoidance of trauma, to the absolute avoidance of smoking, to the continued efforts to develop the collateral circulation by exercising and possibly to the employment of agents which may slow the progression of intravascular thrombosis, specifically antiplatelet substances.

CONCLUSIONS

Lumbar sympathectomy has a definite role in the total care of patients with ischemic feet, whether or not they have diabetes. Results are satisfactory in one-half to two-thirds of patients for the relief of ischemic pain at rest, and healing of ischemic ulcers. Avoidance of amputation for limited periods of time in patients who do not have extensive gangrene beyond the toes can be expected. The site of amputation, whether above or below the knee or at a transmetatarsal site, is not clearly influenced by sympathectomy.

The wholesale performance of lumbar sympathectomy for the relief of claudication is not justified in the light of the natural history of intermittent claudication. Favorable indices for success are angiographic findings showing distal involvement, ankle brachial pressure ratios demonstrating a rich collateral circulation, and lack of extensive gangrene. Prophylactic sympathectomy to improve the results of arterial reconstruction cannot be justified on the basis of available data.

Surgically, lumbar sympathectomy probably should encompass the lowermost preganglionic fiber, the entire lumbar ganglionated chain with ramisectomy of the postganglionic and attached splanchnic fibers with removal of the chain at least to the crossing of the iliac vessels. How to deal with intermediary ganglia, and what role periarterial

stripping of the iliac artery or excision of the sacral sympathetic chain might play are not clear. Frozen section study of the chain at the time of removal is strongly recommended. The problem of postoperative neuropathy for sympathectomy pain is not entirely resolved.

Prospective controlled studies are required to more fully evaluate the results in end stage arterial occlusive disease, the role of predictive tests, and the role of the extent of sympathectomy in achieving more predictable results.

The use of predictive tests which depend upon uncovering vasospasm have not been reliable. The concept of the determination of vascular resistance of the foot is interesting and merits additional observation. Phenol sympathectomy, not generally performed, is a challenging concept.

REFERENCES

1. Berardi, R. S., and Siroospour, D.: Lumbar sympathectomy in the treatment of peripheral vascular occlusive disease. Am. J. Surg., *130*:309–314, 1975.
2. De Bakey, M. E., Burch, G., Ray, T., et al.: The "borrowing lending hemodynamic phenomenon (Hemoneta Kinesia)" and its therapeutic application in peripheral vascular disturbances. Ann. Surg., *126*:850, 1947.
3. De Bakey, M. E., Creech, O., and Woodhall, J. P.: Evaluation of sympathectomy in peripheral vascular disease. J. A. M. A., *144*:1227, 1950.
4. De Takats, G., Fowler, E. F., Jordan, P., et al.: Sympathectomy for treatment of peripheral vascular sclerosis. J. A. M. A., *131*:495, 1946.
5. Ewing, M.: The history of lumbar sympathectomy. Surgery, *70*:790–796, 1971.
6. Folse, R., Mack, R. M., and Cantrell, J. R.: Alterations in femoral blood flow and resistance with and without arterial occlusion by dye dilution technique. Ann. Surg., *162*:873–880, 1965.
7. Friedman, S. A., Frieberg, P., and Colton, J.: Vasomotor tone in diabetic neuropathy. Ann. Intern. Med., *77*:353–356, 1972.
8. Haimovici, H., Steinman, C., Karson, H. I.: Evaluation of Lumbar Sympathectomy. J. A. M. A., *89*:1089–1095, 1964.
9. Imparato, A. M., Kim, G. E., Madayag, M., et al.: Angiographic criteria for successful tibial arterial reconstructions. Surgery, *75*:830–838, 1973.
10. Kim, G. E., Ibrahim, I. M., and Imparato, A. M.: Lumbar sympathectomy in end stage arterial occlusive disease. Ann. Surg., *183*:157–160, 1976.
11. Mackenzie, D. C., and Loewenthal, J.: Lumbar sympathectomy and claudication distance. Surg. Gynecol. Obstet., *115*:303–311, 1962.
12. Mannick, J. A., Jackson, B. T., Coffman, J. O., et al.: Success: of bypass grafts in patients with isolated popliteal artery segments. Surgery, *61*:17, 1967.
13. Monro, P. A. G., Sympathectomy: An Anatomical and Physiological Study with Clinical Applications. London, Oxford University Press, 1959.
14. Moore, W. S., and Hall, A. D.: Effects of lumbar sympathectomy on skin capillary blood flow in arterial occlusive disease. J. Surg. Res., *14*:151–157, 1973.
15. Pick, J.: The identification of sympathetic segments. Ann. Surg., *145*:355–364, 1957.
16. Pick, J.: The Autonomic Nervous System. Philadelphia, J. B. Lippincott, 1970.
17. Raskin, N. H., Levinson, S. A., Hoffman, P. M., et al.: Postsympathectomy neuralgia: Amelioration with diphenylhudantoin and carbamazepine. Am. Jour. Surg., *128*:75–78, 1974.
18. Shanik, G. D., Ford, J., Hayes, A. C., et al.: Pedal vasomotor tone following aortofemoral reconstructions: a randomized study of concomitant lumbar sympathectomy. Ann. Surg., *183*:136–138, 1976.
19. Shaw, S. S., Austen, G. W., and Stipa, S.: A ten year study of the effect of lumbar sympathectomy on the peripheral circulation of patients with arteriosclerotic occlusive disease. Surg. Gynecol. Obstet., *119*:486–496, 1964.
20. Smith, R. B., Dratz, A. F., Coberly, J. C., et al.: Effect of lumbar sympathectomy on muscle blood flow in advanced occlusive vascular disease. Am. Surg.,*37*:247–251, 1971.
21. Szilagyi, E. D., Smith, R. F., Scerpilla, J. R., et al.: Lumbar sympathectomy: Current role in the treatment of arteriosclerotic occlusive disease. Arch. Surg., *95*:753–761, 1967.

22. Walker, P. M., Key, J. A., MacKay, I. M., et al.: Phenol sympathectomy for vascular occlusive disease. Surg. Gynecol. Obstet., *146*:741–744, 1978.
23. Weismann, R. E., and Upson, J. E.: Use of lumbar sympathectomy as an adjunct to reconstructive arterial surgery. Ann. Surg., *154*:788–790, 1961.
24. Webber, R. H.: An analysis of the cross communications between the sympathetic trunks in the lumbar region in man. Ann. Surg., *145*:365–370, 1957.
25. Wright, C. J., and Cousins, M. J.: Blood flow distribution in the human by following epidural sympathetic blockade. Arch. Surg., *105*:334:337, 1972.
26. Yao, J. S. T., and Bergan, J. J.: Predictability of vascular reactivity relative to sympathetic ablation. Arch. Surg., *107*:676–680, 1973.

Department of Surgery
New York University Medical School
550 First Avenue
New York, New York 10016

The Surgery of Varicose Veins

*Chilton Crane, M.D.**

Degenerative changes in the superficial leg veins in the form of dilatation, sacculation, and the tortuosity that follows lengthening rank among the commonest afflictions of American adults. Such varicosities are estimated to occur in perhaps 15 per cent of the adult population.[1]

ETIOLOGY OF VARICOSE VEINS

A logical presentation of steps in treatment must rest on a solid consensus as to first causes together with the patterns of disturbed venous anatomy and physiology that follow.

While decades of debate leave unsettled the question of firm linkage to hereditary traits,[2] most surgeons from their own history taking come to believe that the 15 per cent incidence of varicose veins is concentrated in some small fraction of family lines.

More important, there is universal agreement that functional failure of venous valves is the central pathologic fault. This is seen rarely in the congenital absence of the top greater saphenous valve at the saphenofemoral junction. Much more common is the fibrous contracture of deep venous and communicating venous valve cusps that follows the healing of thrombophlebitis. Most commonly valve failure results from the water hammer effects on venous walls below the inguinal ligament as innumerable downthrusts from sudden increases in abdominal pressure stretch apart valve commissures to produce incompetence. This mechanism sets off a self-perpetuating sequence whereby venous dilatation provokes valve leakage which in turn promotes dilatation of the venous segment just below together with its valveless branches.

Since the common iliac and caval veins are valveless and only one third of external iliac veins contains a protective valve, the topmost

*Emeritus Professor of Surgery, Peter Bent Brigham Hospital, Harvard Medical School, Boston; Chief of Surgery, Veterans Administration Hospital, West Roxbury, Massachusetts

valves in the superficial femoral, greater, and lesser saphenous veins first defend all the leg veins against the sharp rises in intraabdominal pressure that follow coughing, lifting, and straining. The strong circumferential investment of the deep fascia, the vascular sheaths, and the surrounding muscles reinforce the thin walled deep veins from the bursting effects of downward pressure. It is rare to find on phlebography dilatation and valve incompetence of the deep venous system in the absence of prior thrombophlebitis.

Conversely, the greater and lesser saphenous veins with their thick muscular media and well developed elastica, even without external support, are generally fit to tolerate pressures at the ankle on straining while standing which have been measured at 200 mm of mercury.[1]

Again some process of preselection seems at work. Many people have occupations that involve standing, lifting, and straining, but varicose veins develop in relatively few of them. The possibility of congenital defects in vein walls or venous valves comes again to the forefront.

ANATOMIC PATTERNS OF VENOUS VALVE INCOMPETENCE

In the diagnosis and proper treatment of varicose veins it is necessary to picture the four most commonly encountered anatomic patterns.

Group I

In this most basic pattern several of the topmost valves in the greater, far less commonly in the lesser, occasionally in both saphenous systems, are incompetent, allowing downflow on standing or straining. Thus the upper segments of the vein involved will be dilated and perhaps sacculated, as will the branches entering these segments. At times a major branch such as an anterior or accessory saphenous vein may show marked varicose change whereas the greater saphenous trunk below remains normal in caliber and function. As time goes by, the entire saphenous vein, together with many branches, may become sequentially involved all the way down to the ankle.

Group II

Here the proximal greater and lesser saphenous veins are virtually normal but varices, at times grapelike in size, are found in the calf or low thigh. Clearly one or more incompetent communicating veins have been transmitting the high deep vein pressures on exertion through "high pressure leaks" into the saphenous system. The first place to look for such incompetence is just below the adductor canal in the thigh. Often the finger tip can detect the telltale hole in the deep fascia at this level; or a venous tourniquet applied to the elevated drained out leg just below but not above this point, will prevent down

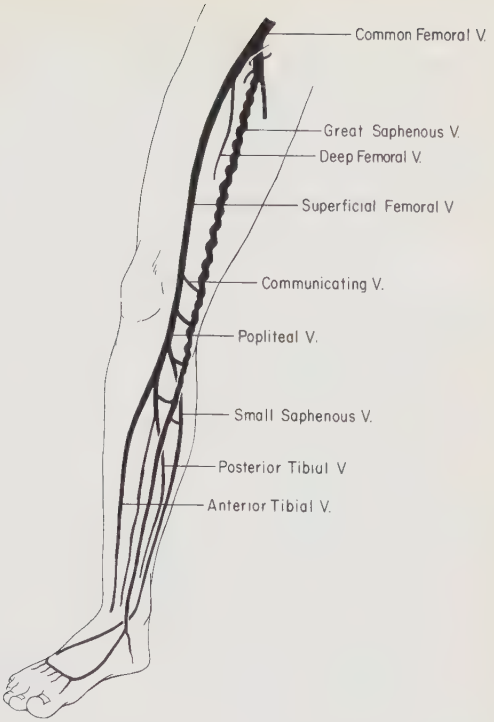


Figure 1. Incompetent high great saphenous veins are demonstrated.

filling of the varices on standing; or a Doppler sensing crystal properly placed will report the rhythmic outrush of blood as the patient does heel raising while standing.

The second place to look for such incompetence is just below the knee medially, where a very constant venous loop joins the greater and lesser saphenous veins and where a major communicating vein "blow-out" may be found.

It is widely recognized that the communicating veins between the saphenous and the deep system or between venous loops and the deep system are most numerous near the ankle and in the lower medial part of the leg, the most typical site for venous ulcer.

Group III

The third pattern is seen where greater or lesser saphenous vein incompetence from the top down coexists with incompetent calf communicating veins. Here the precise sequence of etiologic events is uncertain, although it seems likely that saphenous vein incompetence produced rather than resulted from, communicating vein incompetence.

Group IV

The fourth pattern, most variegated in presentation, most important to recognize and the most difficult to treat, is defined by its one common denominator: prior deep thrombophlebitis resulting in de-

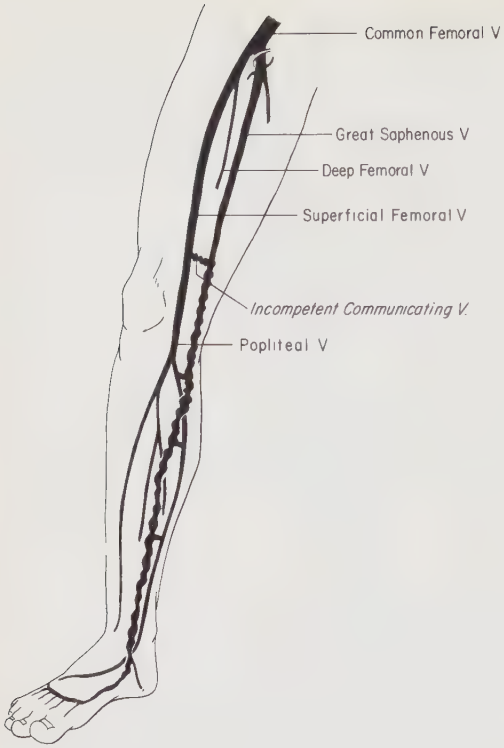


Figure 2. Incompetent communicating veins only are shown.

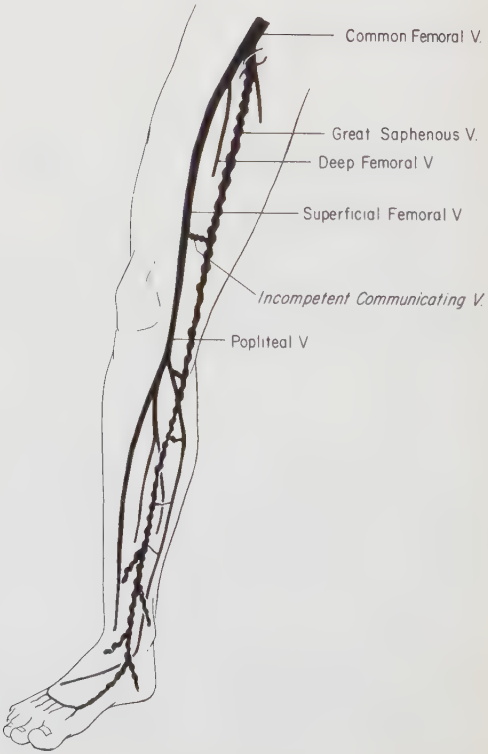


Figure 3. Incompetence of the great saphenous vein and its communicating veins is shown.

struction of venous valves and obstruction of deep veins. In this situation the varicose veins are secondary, by one of two mechanisms. The first is the dilatation of the saphenous system resulting from the enhanced need for collateral flow. The second and far more important is the destruction of communicating vein valves by the phlebitic process or by "blowout" as deep venous pressures mount. One frequent feature of these varicose veins is their wide scatter over the leg outside the usual greater or lesser saphenous distributions.

Any clear history of past deep venous thrombosis is absent in perhaps 60 per cent of such patients. The clinical hallmark is chronic leg edema measurable day after day as a maximum calf circumference exceeding that of the normal leg by 1 cm or more. Secondary features are areas of skin pigmentation and subcutaneous tissue fibrosis due to stasis more extensive and encircling than those seen in groups I, II, and III.

When doubt exists as to the presence of the postphlebitic syndrome, usually because measureable swelling is minimal, or because bilateral involvement negates comparative measurement, phlebography is indicated. Here one looks for deep vein obstructions, intraluminal filling defects or cutoffs, evidence of deep vein valve destruction, and outflow of dye through leaking communicating veins.

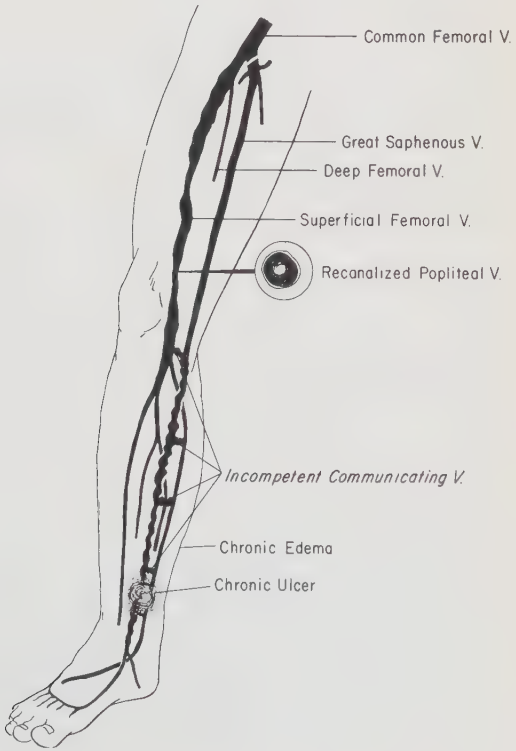


Figure 4. The "post-phlebitic" syndrome is diagrammed.

MEASUREMENTS OF VENOUS PRESSURE IN THE FOUR GROUPS

Before varicose vein surgery can be planned and its outcome predicted, it is essential that the venous pressure abnormalities characteristic of each group be well understood.

When a normal individual stands still, the venous pressure in a dorsal foot vein equals that of a column of blood rising to the right heart, an approximation of arterial pressure. Bernand and Browse[3] have shown the effects of exercise and venous surgery on pressures in the foot veins. In the normal individual after heel raising, pressure in the foot veins drops by 68 per cent of the resting, standing pressure. The calf muscle pump, contracting on the deep veins inside the fascial sleeve, suctions blood through the communicating veins to reduce saphenous pressures at the foot by that amount.

Group I and group III patients who start off with mean pressure falls on exercise of around 40 per cent and 31 per cent respectively, can have these raised by greater or lesser saphenous vein stripping to mean levels of 55 per cent and 54 per cent respectively, thus reapproaching the normal state.

The group II patients, starting off lower at 26 per cent, were raised by saphenous stripping and communicating vein ligation to only 39 per cent. This relatively poor result strongly suggests that many of these patients had deep vein disease with valve injury not diagnosed on the preoperative phlebogram.

It is not surprising that the 40 patients in group IV, in whom damaged deep veins were diagnosed phlebographically, started off with mean falls of only 17 per cent. Combinations of saphenous stripping and communicating vein interruptions improved the percent pressure drop only to 30 per cent.

These unpromising falls in foot vein pressures following venous surgery in group IV, as contrasted with comparable falls in groups I and III, are directly reflected in the results of the operative treatment of stasis ulcer in these groups. In 41 patients with venous ulceration studied preoperatively with phlebography and treated by ligation of calf perforators, recurrent ulcer developed within five years in all of the 23 patients with prior deep venous thrombosis and in only one of 17 patients with normal deep veins.[4]

APPROACH TO THE PATIENT WITH VARICOSE VEINS

The question of first importance in history taking therefore seeks valid evidence for prior deep venous thrombosis. Most casual diagnoses made in a doctor's office and not confirmed by swelling of the leg, hospitalization, anticoagulation, or pulmonary embolism will involve superficial phlebitis, transient edema of the ankle, cellulitis, cramps in the calf muscles or strains — symptoms of no true relevance. The history of acute and continued swelling of one calf or the

whole leg, causing limp and disability, and treated by hospitalization, is usually conclusive.

The second question asks about the complications of varicose veins: ulceration, dermatitis, superficial thrombophlebitis, or hemorrhage. Any of these tend to justify active treatment.

The third question brings out the details of any prior therapy: sclerosing injections, ligations or strippings, anticoagulation, or, rarely, caval ligation or clipping. The history of prior injection therapy warns of difficulties in stripping and the history of prior saphenous stripping directs special attention to residual venous channels at and about the sapheno-femoral junction, and to sites of incompetence of communicating veins.

The cardinal rules for the resection or disconnection of varicose veins are two: the venous segments involved must be demonstrably incompetent, that is, they permit reflux downward and away from the right heart on standing; and they must be sufficiently dilated, tortuous, or sacculated as to be clearly pathologic.

Because the superficial leg veins of patients may be needed at some future time for transfer into obstructed arterial systems, there is no excuse for the inclusion in stripping procedures of virtually normal venous segments. Since varicose veins carry virtually no threat to life or health, it is not justifiable economically or by risk-reward ratio to strip out under general or spinal anesthesia minimally dilated venous trunks.

Thus, lesser saphenous veins, except in the 10 to 15 per cent of cases in which they are involved, or the opposite saphenous vein, when showing only minimal varicose change, should not be stripped out, either for the sake of completeness of procedure or in the name of prophylaxis.

The indications for surgery, then, and the extent thereof, are narrowed down to the resection or disconnection of incompetent and clearly pathologic venous segments.

WHAT SURGERY IN WHAT GROUPS?

In patients in group I greater saphenous stripping should be carried from the lowermost normal venous segment (often none above the ankle), to the sapheno-femoral junction. In some of these patients it may be possible to preserve the lower one third or half of the saphenous vein.

In group II patients, the saphenous trunk in the thigh may be of normal caliber and quite competent; if so, this segment should be preserved. The calf varices should be dissected to their junction with the incompetent communicating veins which are ligated and divided at the level of the deep fascia. The varices themselves are frequently so large, tortuous and sacculated as to be unstrippable. Their complete resection through multiple short incisions is indicated.

In group III patients, complete saphenous stripping with the si-

multaneous disruption of those incompetent communicating veins that form direct T-junctions with the saphenous vein is indicated. Here a special effort is indicated to identify and mark incompetent perforators preoperatively and to note during the stripping those that lie outside the stripping tract. These will require dissection through individual short incisions.

In group IV patients, the first presumption is that conservative therapy with a program of lifelong compression and some compromise between life style and gravity will generally outperform most of the many operations that have been advised. It is noteworthy that virtually all surgeons who are enthusiastic about various operative approaches to the postphlebitic syndrome urge their patients to wear elastic stockings forever postoperatively.

SURGERY IN THE POSTPHLEBITIC STATE

There, are, however, a few situations in which surgery can help the varicose veins and stasis ulcers that follow deep thrombophlebitis.

At times a dilated, sacculated saphenous trunk can be felt with the finger tips as a deep gorge through the indurated, edematous subcutaneous tissue. This is brought out more clearly after three or four days of marked elevation of the limb in bed. If such a vein can be stripped out, stasis changes will lessen.

When an intractable ulcer just above the medial malleolus sits directly astride such a vein, saphenous stripping through the tissues under the ulcer is very worthwhile.

When a recurrent ulcer with relatively healthy tissues around it is found in this location long after saphenous stripping, excision of the ulcer down to and including the deep fascia, with ligation of the incompetent communicating veins beneath, and local skin grafting, may produce a long lasting ulcer cure, provided that leg edema is subsequently well controlled by elastic compression. This procedure requires three weeks of strict bed rest in the hospital, with very gradual lowering of the limb to avoid venous congestion of the graft and its rapid loss.

Selected patients with recurrent ulceration after prior saphenous stripping but with much pigmentation and brawny induration over the medial and lateral aspects of the lower leg, may have relatively normal skin and subcutaneous tissues over the calf posteriorly extending well down over the Achilles tendon. Phlebography may show many incompetent perforating veins.

Here the procedure described by Rob may be useful. A midline incision from above midcalf to just above the os calcis is deepened through the deep fascia. The subfascial plane is developed with Deaver retractors to the tibia medially and the fibula laterally to expose and permit division of all perforating veins.

Unlike the sloughs and infections that often follow medial and lateral incisions made through a densely fibrotic tissue, these posterior incisions tend to heal well.

TECHNICAL POINTS IN SURGERY OF THE VARICOSE VEINS

Marking the Varices

There is little use in marking with various dyes most of the surface veins of the leg. With the patient standing the variceal pattern should be studied and the key channels palpated to detect the telltale grooves in the fat which can be felt equally well the next day on the operating table. It is critically important, however, to mark the sites of all demonstrable perforators, preferably with tiny, shallow, needle scratches. It is important to note accessory or bifid saphenous veins.

The Groin Incision

The oblique groin incision is almost invariably made too low so that forceful upward retraction is needed to properly expose the sapheno-femoral junction and the usual four saphenous branches. The saphenous vein should be ligated exactly flush with the common femoral trunk after its clear exposure just above and below.

The Stripping Tract

The act of stripping out the saphenous vein is a traumatic one. Postoperative tissue reaction can be reduced by using lesser sized stripping heads, and where observation of saphenous connecting veins is unnecessary, by the tight wrapping of the leg over the stripper by Ace bandages before stripping to reduce bleeding.

An infected stripping tract is a disaster. When open ulcers, however clean, are present it is well to wall them off, and strip out the vein from an incision above the ulcer before stripping out the segment below.

Reducing Blood Loss

Most varicose vein operations result in minimal blood loss. In recurrent cases or where varices are large and fragile, potentially larger operative losses can be greatly reduced by placing the patient in a steep head down position.

Use of the Stripper

When the stripper blocks in a venous saccule several maneuvers may be useful. The skin just above the site of block is stretched superiorly and perhaps sideways to alter the configuration of the underlying saccule as new passes are made; or the head of the stripper can be massaged through the skin to take a slightly new course just as the obstruction is reached. Next the stripper is withdrawn and the distal 2 cm section of the plastic cable is slightly bent. Following reintroduction, slow rotation of the proximal end of the cable may permit the eccentric course of the head to negotiate the crooked channel. When all else fails, a second stripper is passed down from above. This may pass through all the way to the ankle or a single cutdown may serve to pick up both strippers.

STRIPPING THE ACUTELY THROMBOSED SAPHENOUS VEIN

When superficial thrombophlebitis ascends into the thigh, prompt high ligation and division of the saphenous vein is indicated. Large clotted varices are often present about the high medial calf. The stripper can be passed downward through thrombus until it blocks. Through several short incisions all varices can be dissected and all clot removed. Remaining segments suitable for stripping can then be removed.

In this setting high ligation alone is often done. While this prevents the rare pulmonary embolus from this source, it leaves the patient with lumpy, painful thrombi that remain in place for weeks and the expectation of saphenous stripping at a later date.

Parenthetically it should be noted that when a normal saphenous vein is found to be thrombosed, it suggests silent deep venous thrombosis propagating through a communicating vein into the saphenous vein, a visceral cancer, vasculitis, or Burger's disease.

OPERATIONS FOR RECURRENT VARICES

When redissecting the saphenofemoral junction in search of inadequate ligations there (57 per cent of cases),[6] it is well to make the incision almost at the inguinal ligament. The dissection comes straight down on the femoral sheath above prior scar and then proceeds to open that sheath over the common femoral vein which is easily dissected downwards. All anterior branches including the saphenous stump, and occasionally the saphenous vein itself or accessory branches are divided and ligated.

SCLEROTHERAPY OF VARICOSE VEINS

Fiscal policy under the National Health Service has made sclerotherapy the premiere mode of treatment in Great Britain. Hobbs[5] personally treated a series of 500 patients, 250 by injection and 250 by surgery, in a random trial conducted between 1966 and 1970. All patients were followed for six years. By the sixth year 82 per cent were cured or improved by surgery, and only 34 per cent by injection treatment.

While few American surgeons advocate sclerotherapy as the primary treatment of major varices, there are five situations in which it is most useful.

First, it may be used for the follow-up management of minor branch varicosities that still fill from below six weeks after stripping procedures, and the control of minor recurrent varices at one or two

year intervals thereafter. A few injections given to possibly one third of cases postoperatively save many patients from multiple extra incisions. Follow-up injections every year or two, if needed, almost invariably eliminate the need for a secondary operation 10 or 15 years later in patients in groups I and III.

Second, sclerotherapy is useful for the immediate obliteration of cosmetically objectionable and occasionally symptomatic, often single, branch varices in young females. It may also be employed for the improved appearance of spidery intradermal telangiectases when they drain into a single 2 to 3 mm, injectable varix. Otherwise these intradermal spidery veins should not be injected as the leakage of any blood from a punctured vein will leave a dime-sized pigment stain for two or three years at the injection site.

In an emergency, sclerotherapy may be important for the immediate control of hemorrhage from a thin walled varix overlain by a tiny acute ulcer. Twenty-three deaths from such lesions were recorded in England and Wales in 1971,[5] mostly in elderly women who bled to death in their sleep. In aged poor risk patients or those refusing surgery, sclerotherapy is a useful office procedure for the management of varicosities causing reflux above chronic ulcers.

TECHNIQUE OF SCLEROTHERAPY

Sodium tetradecyl sulfate (Sotradecal), in 1 and 3 per cent concentrations, is an excellent sclerosing agent most rarely causing either local or systemic complications. Two ml disposable tuberculin syringes bearing 16 mm 25 gauge needles are ideal for these injections.

With the table level a soft venous tourniquet is applied to the leg above the injection site; a loop is left free for easy release. When proper venepuncture is assured by the free aspiration of blood, the tourniquet is released, and the vein is wiped empty by the operator's finger. With the left middle finger of the operator compressing the vein 2.5 cm above the needle, and the index finger 2.5 cm below, 0.5 ml of 3 per cent or 1.5 ml of 1 per cent solution is injected into the empty vein. After waiting about 30 seconds a cotton ball is pressed down over the venous segment and the needle is withdrawn. Firm manual compression is maintained for a minute or two. A band of paper tape one inch wide is applied over the cotton, drawn taut, and stuck down in such a way that pressure is never wholly released.

Each injection provokes an immediate profound local vasospasm in that venous segment. Therefore, when injecting the same vein at two or three levels, one must proceed from above downward. Firm Ace bandage wrapping is used for several days, but the patient may remain ambulatory and go to work.

In these specific areas, sclerotherapy proves to be a most valuable adjunct to the surgery of varicose veins.

REFERENCES

1. Blunoff, R. L., and Johnson, G.: Saphenous vein PO_2 in patients with varices. G. Surg. Res., 23:35, 1977.
2. Burkitt, D. P.: Varicose veins, facts and fantasy. Arch. Surg., 3:1327, 1976.
3. Bernand, K. G., O'Donnell, T. F., Thomas., M. L., et al.: Relative importance of incompetent communicating veins in the production of varicose veins and venous ulcer. Surgery, *82*:9, 1977.
4. Bernard, K. G., O'Donnell, T. F., Thomas, M. L., et al.: Venous ulcers — Surgical treatment. Lancet, *1*:936, 1976.
5. Hobbs, J. T.: Surgery and sclerotherapy in the treatment of varicose veins. Arch. Surg., *109*:793, 1974.
6. Nabatoff, R. A.: Technique of operation upon recurrent varicose veins. Surg. Gynecol. Obstet., *143*:463, 1976.

Peter Bent Brigham Hospital
721 Huntingdon Avenue
Boston, Massachusetts 02115

Management of Arterial Emboli

*Thomas J. Fogarty, M.D.**

Acute arterial occlusion may be a result of in situ arterial thrombosis or embolization from a more proximal source. In the majority of cases, thrombosis and embolization are easily distinguished from one another. Difficulty in making a distinction between the two forms of ischemia occurs in approximately 15 per cent of cases. Confusion as to the exact cause of ischemia may be the result of the absence of an obvious source for the embolus, the absence of heart disease, and slowly progressing signs and symptoms of ischemia. In situations in which careful history and physical examination fail to make a clear-cut distinction, arteriography can be of considerable aid. It is exceedingly important to distinguish between embolization and thrombosis. The natural history of thrombosis and embolization at levels of occlusion are different. In addition, pathologic conditions encountered in the two forms of acute arterial occlusion are very different and surgical techniques to restore blood flow are dissimilar. Extensive reconstruction and time-consuming surgical techniques are often required to assure the viability of tissues in patients with acute thrombosis. Advanced and difficult techniques are rarely needed in patients with acute embolization.

Preoperative Evaluation and Care

The sudden onset of symptoms of ischemia in patients with atrial fibrillation should immediately indicate the diagnosis of an arterial embolus. In the absence of atrial fibrillation or an obvious source for the embolus, acute arterial thrombosis is a more likely diagnosis.[2] Cranley makes the point that pain is the initial sign of an embolus while numbness is the first sign of thrombosis. Independent of the cause of the acute ischemia, the clinical situation represents a medical emergency. The color and temperature of the extremity, the presence or absence of sensation and of sense of position, and the level of motor movement are recorded and related to time of onset. The level and amplitude of pulsations should be recorded by Doppler techniques. In addition to recording

*Staff Surgeon, Sequoia Hospital, Redwood City, California; Private Practitioner, Palo Alto, California

of the signs of acute ischemia, the examination and questioning of the patient should include an investigation into the possible presence of any preexisting arterial insufficiency. Knowledge of the presence of significant atherosclerosis aids in defining the goals of immediate therapy. The advisability of attempting to reconstruct a normal lumen in a chronically diseased vessel following acute arterial occlusion depends on many factors. Extensive ischemia may force urgent surgical intervention under less than optimal medical conditions. In cases of acute arterial thrombosis occurring in a critically ill patient, the use of heparin may be preferable to immediate surgical intervention. Lengthy reconstructive procedures are often best delayed until a complete diagnosis and therapeutic evaluation of the patient has been possible.

In cases of embolic occlusion, significant cardiac disease frequently coexists, and is in fact the most frequent cause of the embolic event. A significant part of preoperative care should be directed to improving myocardial performance. Isolation in the intensive care unit with appropriate monitoring is indicated. The use of digitalis, antiarrhythmic agents, morphine, diuretics, and heparin are drugs basic to the care of the patient presenting with an acute embolus. Heparin is given immediately upon diagnosis in the hope of diminishing the likelihood of repeat embolization. The distal propagation of thrombus is also minimized by the use of heparin.

The frequency and severity of underlying cardiac disease should not be underestimated. After stabilization of cardiac status and management of the acutely ischemic extremity, a careful and thorough cardiac evaluation is in order. Cardiac catheterization and coronary arteriography may be needed in the immediate postoperative period. The potential for coexistent visceral or renal emboli must be borne in mind. Arteriography should be utilized if the clinical presentation causes concern that embolization may occur in areas other than the extremity. In situations in which the diagnosis of embolization to an extremity is clear-cut, preoperative arteriograms are not utilized. In clinical settings in which the distinction between embolization and thrombosis is not clear, arteriography often establishes the diagnosis. An awareness that an aortic dissection may mimic an arterial embolus should be sufficient to avoid confusing the two catastrophic events. Back pain in the thoracic area is uniformly seen in acute dissections and precedes the symptoms of ischemia.

Operability

The primary objective in managing the patient with acute arterial occlusion is to assure survival of the patient. The secondary objective is the attempt at salvaging a functional extremity. Reference to time as a primary determinant of potential for limb salvage persists even in current publications.[1]

In general it is true that longer time intervals of occlusion are associated with greater degrees of ischemia. The pathology and physiology responsible for significant and frequent exceptions to this concept of time as a determinant of operability have been previously stated.[9]

The condition of the extremity at the time the patient is initially seen

is the real determinant of limb salvage. A profound lack of motor activity in association with a doughy consistency of muscle mass is a contraindication to embolectomy. In the presence of advanced ischemia as defined by early rigor, the amputation rate is higher but total salvage of the limb is possible. If one is to operate in the presence of advanced ischemia, potential complications associated with the reestablishment of blood flow to a marginal viable extremity must be anticipated. Coexistent venous thrombosis is often found when early rigor is a presenting finding. The technical aspects of this particular situation demand clamping of the common femoral vein before reestablishment of arterial flow. After successful restoration of arterial flow, venous thrombectomy or generous flushing of the venous efflux is performed.[5]

The sudden return of blood to the heart from an extremity with advanced ischemia can have significant adverse effects.[3,7,8] Pooling of blood in the revascularized extremity in conjunction with the return of acidotic blood to the heart can cause fatal arrhythmia or refractory hypotension. These adverse effects can be avoided by volume replacement and the use of appropriate antiarrhythmic agents.

Operative Technique

The basic concept of the balloon catheter for the performance of embolectomy has not changed since its initial introduction.[6] It should be emphasized that the Fogarty embolectomy catheter was not designed for endarterectomy or arterial dilatation. Figure 1 illustrates the basic concept and use of the catheter. Incisions are made in the artery over the bifurcation so that the common, deep and superficial femoral arteries can be easily cannulated. The femoral incision is usually adequate to remove embolic occlusions in any location. On occasion, the need to selectively cannulate the tibial vessels may necessitate incisions over the distal popliteal artery. As repeatedly emphasized, back bleeding is an unreliable guide to distal patency.[4] Familiarity with the technique, use, and limitations of the instrument best serves to minimize complications.

Postoperative Care

Local care of the involved extremity includes all routine measures employed in elective vascular reconstructive procedures. Heparin therapy is initiated approximately 12 hours postoperatively and continued until the patient is ambulatory. Significant swelling may occur following revascularization. If edema appears to compromise arterial inflow, fasciotomy should be performed.

The need for directing care to the underlying cardiac disease should not be overlooked. After recovery from the acute embolic event, further cardiac studies are often required. Thomas correctly emphasized the potential need for direct cardiac surgery in increasing numbers of these patients.[10]

Conclusion

Acute embolic occlusion occurs in clinical settings associated with significant cardiac disorders. Appropriate use of surgical techniques in

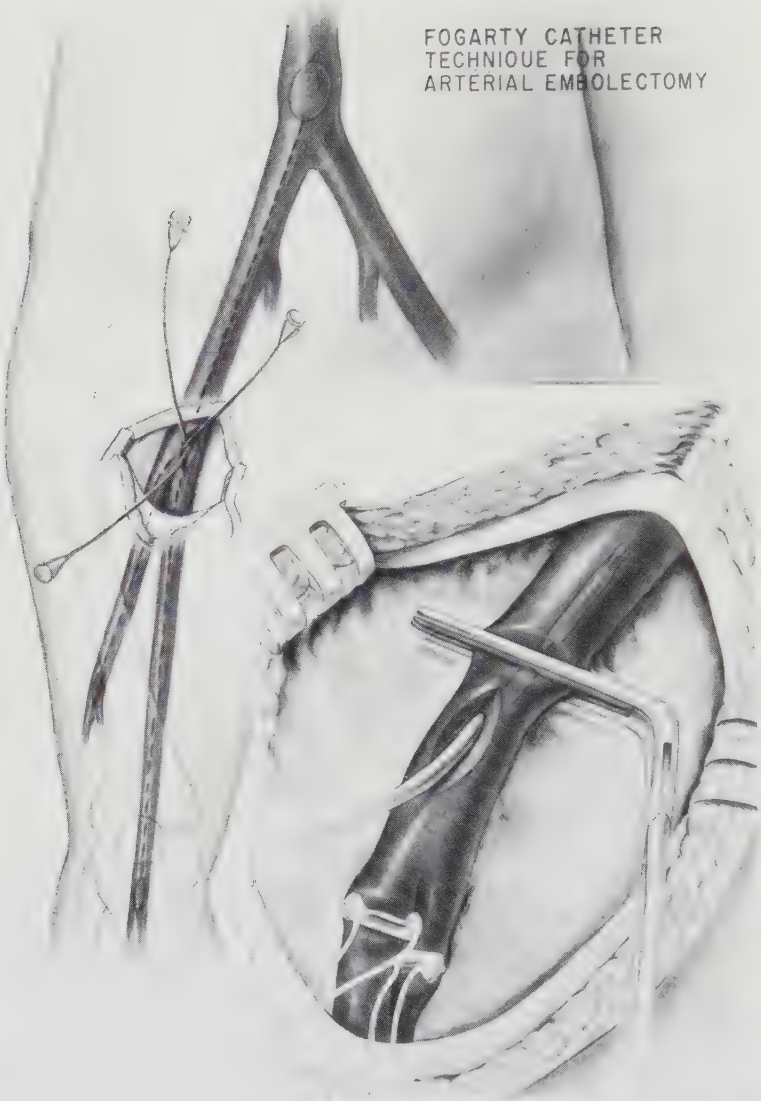


Figure 1. The Fogarty catheter is used to cannulate the common, deep, and superficial femoral arteries to remove embolic occlusions.

conjunction with aggressive medical and surgical management of the underlying cardiac disorder will result in improved morbidity and mortality statistics.

REFERENCES

1. Blaisdell, F. W., Steele, M., and Allen, R. E. .: Management of acute lower extremity arterial ischemia due to embolism and thrombosis. Surgery, *84*:822, 1978.
2. Cranley, John J.: Vascular Surgery. Volume 1. New York, Harper and Row, 1972.
3. Fisher, R. D., Fogarty, T. J., and Morrow, A. G.: Clinical and biochemical observations of the effect of transient femoral artery occlusion in man. Surgery, *68*:323, 1970.
4. Fogarty, T. J.: The balloon catheter in vascular surgery. Rev. Surg., *24*:9, 1967.
5. Fogarty, T. J.: Complications of Arterial Embolectomy. *In* Beebe, H. G., (ed.): Complications in Vascular Surgery. Philadelphia, Lippincott., 1972.
6. Fogarty, T. J., Cranley, J. J., Krause, R. J., et al.: A method for extraction of arterial emboli and thrombi. Surg. Gynecol. Obstet., *116*:241, 1963.
7. Green, R. M., DeWeese, J. A., and Rob, C. G.: Arterial embolectomy before and after the Fogarty catheter. Surgery, *77*:24, 1975.
8. Haimovici, H., Moss, C. M., and Veith, F. J.: Arterial embolectomy revisted. Surgery, *78*:409, 1975.
9. Krause, R. J., Cranley, J. J., Strasser, E. S., et al.: Further experience with a new embolectomy catheter. Surgery, *59*:81, 1966.
10. Thomas, T. V.: Arterial embolectomy: Role of anticoagulants and arteriography, J. Cardiovasc. Surg., *13*:584, 1974.

770 Welch Road
Suite 201
Palo Alto, California 94304

Index

Note: Page numbers of symposium and article titles are given in **boldface** type.

Amaurosis fugax, in carotid artery disease, 539
Amputation, lumbar sympathectomy and, 731
Aneurysms, aortic, treatment of, current status, **597–636**
  arterial, peripheral, management of, **693–706**
  false, as complication of vascular surgery, 664–665
  mycotic, in drug addict, 701–702
    primary, 703
  of aorto-iliac system, extraanatomic bypass grafts for, 650–651
  popliteal, 703–704
Angiography, before profundaplasty, 684
Antibiotics, in femoro-popliteal-tibial grafting, 589
  prophylactic, in operation for aortic aneurysm, 599
Aorta, aneurysm of, surgical treatment of, **597–636**
  reconstruction of, in aorto-iliac disease, **565–579**
Aortic arch, branches of, and vertebral arteries, atherosclerosis of, surgery for, **669–680**
Aorto-femoral-popliteal reconstruction, simultaneous, 577–578
Aorto-iliac reconstruction, **565–579**
  endarterectomy vs. bypass grafting in, 571–573
Aorto-iliac segment, arteriosclerotic disease of, 565–570
Arteries, aorto-femoral-popliteal, reconstruction of, simultaneous, 577–578
  aorto-iliac, reconstruction of, **565–579**
  carotid, occlusive disease, endarterectomy for, 677–678
    surgery, complications following, 661
      in cerebral ischemia, **539–553**
  celiac, revascularization of, 642
Arteries (*Continued*)
  emboli of, Fogarty catheter for removal of, **749–753**
  femoral, aneurysms of, 694–700
  femoro-popliteal and femoro-tibial, reconstruction of, **581–596**
  femoro-tibial, reconstruction of, for limb salvage, 589–593
  occlusive disease of, surgery for, endarterectomy vs. bypass grafting, 679–680
  proximal extracranial, atherosclerosis of, surgery for, **669–680**
  reconstruction of, complications following, **659–668**
  renal, fibrodysplastic disease of, dilatation for, 561
    reconstruction of, **555–563**
    surgery for, complications of, 661
  vertebral, and aortic arch, atherosclerosis of, surgery for, **669–680**
    endarterectomy for, 678–679
Arteriography, in cerebral ischemia, 543, 552
  in determination of renal artery disease, 557–558
  in intestinal ischemia, 638, 640
Atherosclerosis, progression of, following revascularization procedures, 667–668

Brain, ischemic, carotid surgery for, **539–553**
Bruits, carotid, asymptomatic, management of, 550–552
Bypass grafts, extraanatomic, **649–658**

Catecholamines, and vasoconstriction, 723
Celiac compression syndrome, 642
Claudication, profundaplasty for, 689–690

DOPPLER tests, before lumbar sympathectomy, 726–727
in evaluation of femoro-popliteal-tibial occlusive disease, 582–583
Drug addict, femoral aneurysms in, 701–702

EMBOLISM, arterial, acute, vs. thrombosis, in diagnosis of acute arterial occlusion, 749–750
management of, **749–753**
as complication of lumbar sympathectomy, 729
as indication for endarterectomy, 673–674
intestinal, vs. thrombosis, 638
Endarterectomy, aorto-iliac, 572–574
carotid, 544–550
for visceral ischemia, 641–644

FEET, ischemic, lumbar sympathetctomy for, **719–735**
Fistula, aorto-caval, surgery for, 633
aorto-duodenal, surgery for, 634
Fogarty catheter, for removal of arterial emboli, **749–753**

GRAFTS, aortofemoral, 571–577
aorto-iliac, infected, extraanatomic bypass grafts for, 651
autogenous, in femoro-popliteal-tibial reconstruction, 583–593
bypass, extraanatomic, **649–658**
for cerebrovascular ischemia, 675, 677, 679
Dacron, failure of, in vascular procedures, 664
in intestinal revascularization, 642, 645–646
in repair of aortic aneurysm, 602–632
inclusion technique for, 630–631
saphenous vein, in intestinal revascularization, 645
in profundaplasty, 688

HEMORRHAGE, following arterial reconstruction, 660
Heparin, in profundaplasty, 687
Hypertension, during endarterectomy, treatment of, 545
renal, diagnosis of, intravenous pyelogram for, 555
renin assays in, 556

INFECTION, postoperative, following arterial reconstruction, 660, 665–667

Ischemia, causes of, embolization vs. thrombosis, 749–750
cerebral, arteriography in, 543, 552
carotid surgery for, **539–553**
diagnosis of, carotid phonoangiography in, 544
cerebrovascular, mechanisms of, 672
colorectal, as complication following vascular procedures, 662
in aorto-iliac segment, 565–570
intestinal, surgery for, 641–642, 644–646
tests for, 637–641
of leg, extraanatomic bypass graft for, **649–658**
of lower limb, femoro-popliteal and femoro-tibial reconstruction for, **581–596**
grafting vs. profundaplasty for, 587–588
of spinal cord, as complication following vascular procedures, 663
renal, surgery for, **555–563**
transient cerebral, as warning of stroke, 539
endarterectomy for, 541

KIDNEY, ischemic, renal artery reconstruction for, **555–563**

LABORATORY, vascular, use and abuse of, **707–717**
Leg, ischemic, extraanatomic bypass grafts for, **649–658**
femoro-popliteal and femoro-tibial reconstruction for, **581–596**
revascularization of, profundaplasty for, **681–691**
Leriche syndrome, in aorto-iliac disease, 566
Limb, salvage of, femoro-popliteal grafting for, 584–588
femoro-tibial reconstruction for, 589–593
grafting vs. profundaplasty for, 587–588

NERVES, sympathetic, lumbar, and vasoconstriction, 722–724

OCULOPLETHYSMOGRAPHY, in diagnosis of cerebral ischemia, 544

PARAPLEGIA, after surgery for aortic aneurysm, 628–629
following vascular procedures, 664
Peripheral vascular surgery, symposium on, **537–753**

Phlebography, in diagnosis of varicose veins, 741–742
Phonoangiography, carotid, in diagnosis of cerebral ischemia, 544
Profundaplasty, for revascularization of lower extremities, **681–691**
Prosthesis, Dacron, in aorto-iliac bypass grafting, 574
in intestinal revascularization, 642, 645–646
Pseudoaneurysms, infected, traumatic, 702
Pyelogram, intravenous, in renovascular hypertension, 555

RADIONUCLIDE scans, in cerebral ischemia, 543
Renal. See *Kidney.*
Renin assays, in diagnosis of renovascular hypertension, 556

SAPHENOUS vein, as autograft in femoropopliteal-tibial reconstruction, 583–593
as autograft in revascularization, 558–560
Scans, radionuclide, in cerebral ischemia, 543
Sclerotherapy, for varicose veins, 746–747
Shunting, in endarterectomy, 545
Stroke, prevention of, carotid surgery for, 541–543
Sympathectomy, lumbar, for arterial disease in lower extremities, **719–735**
Sympathetic nerves, lumbar, and vasoconstriction, 722–724

TESTS, Doppler, in diagnosis of femoropopliteal-tibial occlusive disease, 582–583
in postoperative monitoring of bypass graft surgery, 655
noninvasive, for vascular disease, **707–717**
predictive, before lumbar sympathectomy, 725–727
renin, for diagnosis of renal hypertension, 556
Thrombophlebitis, as cause of varicose veins and stasis ulcers, surgery for, 744
Thrombosis, arterial, following operation for vascular reconstruction, 660, 663
Transient ischemic attacks, as indication for endarterectomy, 541
as warning of stroke, 539

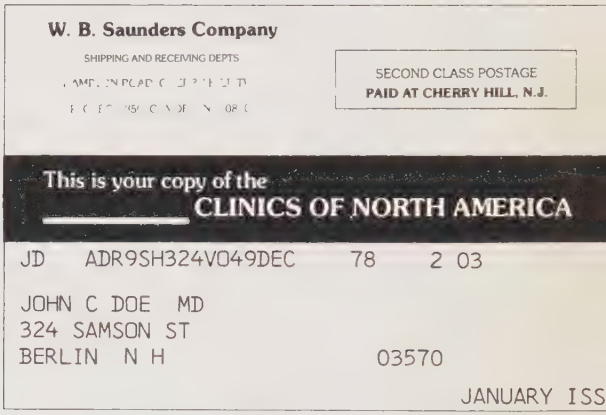
ULCERS, stasis, following thrombophlebitis, surgery for, 744
Ultrasound tests, in diagnosis of peripheral arterial aneurysm, 700

VALVES, venous, incompetence of, in varicose veins, 737–741
Veins, saphenous, as autograft in femoropopliteal-tibial reconstruction, 583–593
as autograft in intestinal revascularization, 642, 645–646
as autograft in kidney revascularization, 558–560
stripping of, for varicose veins, 743–746
varicose, surgery for, **737–748**

Changing Your Address?

Make sure your subscription changes too! When you notify us of your new address, you can help make our job easier by including an exact copy of your Clinics label number with your old address (see illustration below). This number identifies you to our computer system and will speed the processing of your address change. Please be sure this label number accompanies your old address and your corrected address—you can send an old Clinics label with your number on it or just copy it exactly and send it to the address listed below.

We appreciate your help in our attempt to give you continuous coverage. Thank you.


W. B. Saunders Company
SHIPPING AND RECEIVING DEPTS
SECOND CLASS POSTAGE
PAID AT CHERRY HILL, N.J.
This is your copy of the
CLINICS OF NORTH AMERICA
JD ADR9SH324V049DEC 78 2 03
JOHN C DOE MD
324 SAMSON ST
BERLIN N H 03570
JANUARY ISS

Your Clinics Label Number

Copy it exactly or send your label along with your new address to:
W. B. Saunders Company, Dept. C.A.C.,
West Washington Square, Philadelphia, PA 19105.

If you subscribe to more than one Clinic, please include your Clinics label number (or the entire mailing label) for each subscription.

Please allow four to six weeks for delivery of new subscriptions and for processing address changes.

For handy clinical guidance, reach for Saunders Periodicals

Up-to-date information in a clear concise format, that's what you get when you reach for **Saunders Periodicals!**

These useful clinical volumes put the professional information you need right at your fingertips. Each volume in the series contains explicit, in-depth coverage of current topics of interest, plus first hand reports on new developments by leading experts in the field and plenty of fresh practice-applicable advice. So make sure these unmatched clinical references are there when you need them – enter your subscription today! Simply tear off the card below and drop it in the mail.

Changing your address?

Make sure your subscription goes with you. Notify us promptly so your subscription can continue uninterrupted. If available, submit your Clinics mailing label with your address change or copy your Clinics label number exactly in the space provided below. Please allow 4 to 6 weeks for processing address changes.

Canadian Subscribers: Please send your order cards to: W. B. Saunders Company, 1 Goldthorne Avenue, Toronto, Ontario M8Z 5T9 (Prices for these Clinics are 20% higher in Canada.)

Detach and Mail (Printed in USA. Prices are U.S. only and subject to change.)

Office use only. Please don't write here.

TRANS
2
ACT-1
9
ACT-2
TAX CODE
TERMS
SHIP
PRI
MSG

Beginning with the current issue, start my subscription to:

- ☐ **The Dental Clinics** Pub. Quarterly. $20.00 per year.
- ☐ **Human Pathology** – A soft-cover journal. Pub. Bi-monthly. $38.50 per year.
- ☐ **The Medical Clinics** Pub. Bi-monthly. $27.00 per year.
- ☐ **The Nursing Clinics** Pub. Quarterly. $19.00 per year.
- ☐ **The Orthopedic Clinics** Pub. Quarterly. $30.00 per year.
- ☐ **The Otolaryngologic Clinics** Pub. Quarterly. $36.00 per year.
- ☐ **The Pediatric Clinics** Pub. Quarterly. $21.00 per year.
- ☐ **Clinics in Plastic Surgery** Pub. Quarterly. $50.00 per year.
- ☐ **Primary Care: Clinics in Office Practice.** Pub. Quarterly. $25.00 per year.
- ☐ **The Surgical Clinics** Pub. Bi-monthly. $30.00 per year.
- ☐ **The Veterinary Clinics: Small Animal Practice.** Pub. Quarterly. $29.50 per year.

CHANGE OF ADDRESS

I subscribe to: _____

Label No. _____

Old address _____

_____ ZIP _____

Effective _____ / _____ / _____

Send to new address shown below.
Please allow 4 to 6 weeks for processing address changes.

- ☐ Bill me
- ☐ Check enclosed (Saunders pays postage & handling in U.S.)

Please Print

Name _____

Address _____

City _____

State _____ ZIP _____

- ☐ Credit my salesman.

Saunders Periodicals

Simply detach the card below to start your subscription(s). Don't forget! Mail it today!

NO POSTAGE NECESSARY IF MAILED IN THE UNITED STATES

BUSINESS REPLY CARD

FIRST CLASS PERMIT NO. 101 PHILADELPHIA, PA

Postage will be paid by addressee

W.B. Saunders Company
West Washington Square
Philadelphia, PA 19105

CØØJYS

C.U.M.C.
CØØJYS